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The Massachusetts Medical Society

SECTION OF MEDICINE

WEDNESDAY MORNING, JUNE 6, 1928

THE Section of Medicine of the Massachusetts Medical Society, held in the Large Dining Room of the Hotel Bancroft, Worcester, Massachusetts, convened at nine-twenty o'clock, Dr. William R. Ohler, of Boston, Chairman, presiding.

CHAIRMAN OHLER: The first speaker and the last speaker haven't yet arrived, so we will begin with the second speaker on the program, Dr. Albee.

I should like to say at the start that the plan of the meeting is to have the speakers read their papers as listed on the program, and then hear the discussion from the men selected to discuss, as given on the program, then go to the next paper. At the end of the whole session we hope that there will be sufficient time to have a good, general discussion of the whole four papers.

In the absence of Dr. Adams, who was to open the meeting with a paper, I am going to call on Dr. George M. Albee, of Worcester, who will read a paper entitled "Care of the Heart in Pneumonia." Dr. Albee. (Applause.)

THE CARE OF THE HEART IN PNEUMONIA*

BY GEORGE M. ALBEE, M.D.†

MUCH has been written and many different points of view have been expressed concerning the treatment of the heart in pneumonia. This very conflict in the point of view must make one think that no great advantages exist in one method as compared with another in the care of the heart during an active pneumonia. If the problem were simple and the solution evident, certainly by now everyone would be in agreement as to the proper procedures. The main contention that has arisen is whether digitalis should be used, inasmuch as this is the one drug that we know acts favorably in heart failure.

At the outset one must differentiate a toxemia with its prostration, cyanosis, rapid pulse and breathing, and a failing heart with passive congestion engorged veins and liver and edema. A patient who has had mitral stenosis and auricular fibrillation for many years and has shown increasing evidences of congestive heart failure, such as dyspnoea, cyanosis and pitting edema is frequently improved by digitalis, but finally dies of what we call circulatory failure. On the other hand, it cannot be said that a patient who bleeds to death from a ruptured extra-uterine pregnancy has died of circulatory failure, although the pulse rate had accelerated to a great degree. The same is true of patients who die of puerperal sepsis, although in both of these two instances final exitus does occur when the heart is no longer able to supply oxygen to the tissues of the body. In other words we are accustomed to consider the time of exitus as synchronizing with the cessation of the heart beat, but that does not mean that the heart is particularly abnormal in this process. It is quite evident that digitalis would be ineffective in warding off a fatality in the case of tetanus poisoning, as death does not occur from heart failure but from toxemia. With this point of view in mind, let us consider the problem as it exists in pneumonia.

Not long ago Porter and Newburgh in some experiments on dogs showed that when the animal was dying of pneumonia, if the heart were excised and perfused with normal blood, rather than with blood that contained the toxins of the pneumococcus, it would contract normally. That is, the heart muscle was ready to do its work, but was being constantly poisoned by the product of the infection. If we assume that the patient who comes down with pneumonia, had to begin with a normal heart, there is little reason to think that the heart will make other than a normal response to the infection, with the single exception that in some cases an abnormal rhythm may develop, such as auricular fibrillation. During pneumonia the heart rate is rapid, due to the general toxemia and the fever. There are also profound changes in the vasomotor apparatus due to the toxic action on the higher centers which effect results in an atonic peripheral vascular state. There is cyanosis, but not for the same reason that it is present in patients with mitral stenosis. The cyanosis is due partly to a direct change in the character of the

*Read before the Annual Meeting of the Massachusetts Medical Society at Worcester, June 6, 1928.

†For record and address of author see "This Week's Issue," page 445.

blood as a result of the toxins of pneumonia and to a lack of aeration of the blood as it circulates through the lungs. This lack of aeration is a result of both the actual consolidation of lung tissue and the edema that exists in the alveoli and bronchioles of the lungs. These changes prevent the inspired air from coming into intimate contact with the circulating blood. A therapeutic inference that may be drawn from this consideration is that oxygen in higher concentration than exists in the air might prove valuable in diminishing the cyanosis. In this way, even with the restricted exposure of the blood to the air, a larger amount of oxygen would be absorbed. Barach in N. Y. and others have noted the improvement in cyanosis in pneumonia patients when they inhale 50% oxygen by means of an oxygen tent or chamber. No such direct or specific results on the cyanosis have been noted following the use of digitalis.

When patients manifest signs of congestive heart failure, such as pitting edema, engorgement of the veins and liver, hydrothorax etc., we all know that digitalis may be helpful. These signs are generally lacking in pneumonia. A further important point is the fact that the heart is essentially regular in pneumonia, and the degree of slowing produced by digitalis on the regular heart is not striking. The marked slowing of the heart rate that digitalis produces in general practice occurs when auricular fibrillation is present. This condition develops in approximately 5%—8% of the cases of pneumonia, and here digitalis may be of great value and occasionally life-saving. For when this complication occurs, the heart rate becomes very rapid and irregular. There develops a marked pulse deficit and a specific embarrassment of the circulation as a direct result of this new complication. Digitalis can and does improve quite decidedly this particular feature in pneumonia patients. The evidence for the value of digitalis in pneumonia when the heart is regular is very doubtful. In the last analysis the only criterion is a lowered mortality rate. No controlled group has ever been published that would lend support to the view that digitalis has diminished the mortality of pneumonia.

It was thought some years ago that digitalis had no action in the presence of fever. This has been disproved by Dr. Cohn of the Rockefeller Hospital and his associates. He has shown that digitalis produces the same changes in the electrocardiogram both experimentally and in the human, during pneumonia as it does normally. He further has brought forth evidence that during pneumonia, if digitalis is given, the heart is apt to be slightly smaller, i. e. it is in a somewhat better state of tonus. We cannot, however, conclude from these observations that the changes above noted are necessarily beneficial, although they are suggestively so. We do know, therefore, that digitalis does act in the presence of pneumonia, but how desirable that effect actually is, remains uncertain.

There has been some very recent work done in Nashville by T. R. Harrison and his associates, which, if true, has a bearing on this problem. He found experimentally that digitalis actually diminishes to a slight extent the output of the heart, and he suggested that it might be harmful in certain conditions like pneumonia. I do not think that either his work or his inference has had general acceptance as yet but it is at least something we must think about.

There are many men of excellent repute who give digitalis in full doses as a routine to every patient with pneumonia. There are others of equally sound judgment who do not give it at all. There are finally those who give digitalis only when they think the heart needs it. This is a very loose expression and probably means only when the patient is very sick, which all pneumonia patients are shortly before they die. If it meant that digitalis were given only when auricular fibrillation developed, their position would be logical, but that generally is not the case. It is presupposed in this discussion that the patient did not have congestive failure to begin with, for if he had, it would be acknowledged by all that digitalis is highly desirable. One may readily see from the above remarks how divergent are the current views concerning the proper use of digitalis in pneumonia. Much more work, both experimental and clinical, will be needed before a proper solution of this problem is obtained.

Apart from digitalis, there is much in the treatment of pneumonia that tends to help the heart and the circulation. The general care of the patient, the relief of the abdominal distention by oil and enemas, the relief of pain and cough by morphine, the specific serum therapy and the like, in so far as they diminish the toxemia and the bacteremia will directly help the heart. Morphine will often change a small thready pulse to a fuller one, and will diminish the respiratory rate, especially if there has been pleural pain. The patient will breathe deeper and aerate better, thereby requiring fewer breaths than when the pain made him take short shallow ones. The diminished muscular action and the sleep that follows the relief from pain conserves the heart muscle.

There is one therapeutic measure that deserves special mention, as it has a direct bearing on the circulation. That is bleeding. There are occasional instances in pneumonia when there is marked cyanosis and pulmonary edema. Such patients not only have consolidation of one part of the lung, but also have moist bubbling râles indicating an edematous state in other portions of the lungs. Here removing 400cc.-500cc. of blood from one of the veins of the arm has at times been attended by marked improvement in the clinical condition of the patient. When such an effect is produced, it probably results from

diminishing the load of venous blood in the pulmonary circulation. In selected cases, therefore, bleeding is a valuable aid in our therapy. Caffeine sodium benzoate as a respiratory stimulant at times has a slight beneficial effect in some of the toxic cases with marked dyspnoea. Adrenalin helps at times when the blood pressure is low.

The points that I wish to make are, first—that we should differentiate toxemia, with perhaps bacteremia, and its effect on the heart and vasomotor apparatus and the higher centers, and a failing myocardium with congestive failure; second—digitalis is called for when there is congestive failure, with edema and engorged veins and liver, or when there is fibrillation with a pulse deficit and an impending failure. Cecil of N. Y. says there is one fact the significance of which all students of pneumonia are agreed upon, namely, that fatal pneumonia is usually a septic pneumonia. There are pneumococci in the circulating blood. And the patient dies of a vasomotor paralysis. And digitalis will not help him. A murmur or a valvular lesion does not call for digitalis during pneumonia any more than it does at any time.

We could sum the whole thing up in one sentence—Let the heart alone, if it is doing its work, that is, if there is no edema and no engorgement of the veins.

CHAIRMAN OHLER: The discussion on this paper will be opened by Dr. Burton E. Hamilton, of Boston.

DISCUSSION

DR. BURTON E. HAMILTON: *Mr. Chairman and Gentlemen*—I agree with the points that Dr. Albee has made. My discussion will simply emphasize a few of them. In my opinion, Dr. Albee did well to emphasize the importance of differentiating heart failure and circulatory failure in all seriously sick patients. This distinction is fundamental to sensible treatment. Where the heart is failing to do its work, the veins will be generally congested. Where, however, there is cyanosis, breathlessness, rapid heart, due to circulatory failure, not to heart failure, the veins are not congested.

Heart failure in pneumonia does occur, but it is very rare. To be sure, at autopsy in death from pneumonia, one sometimes finds the right heart dilated and, of course, congestion in the lungs. But the congestion in the lungs is usually sharply localized and there is seldom general systemic venous congestion.

The matter of routine digitalization in pneumonia will never be settled until someone pairs a series of cases, digitalizes one and does not digitalize the other, and follows a sufficiently large series of such pairs. We attempted to do that at the Boston City Hospital some years ago, but were unable to follow the work through. Dr. White and Dr. Burrage reported the results of an investigation of records of a long series of pneumonia cases in the Massachusetts General Hospital some time ago, and they found that there was very little difference in the death rate between the patients who had had digitalis and those who had not. But routine digitalization is still a matter of opinion. In Dr. Albee's opinion and in my opinion, routine digitalization is not indicated. We use it, as he said, where there is auricular fibrillation. This occurs in a small percentage of pneumonia cases, as it does in any disease or condition where there is a strain on the heart. Old hearts and otherwise damaged hearts may develop fibrillation at any time under strain. It is a complication; it demands treatment. It is not a matter of opinion whether digitalis is good for auricular fibrillation; we all know that it is. We also digitalize patients in pneumonia if there is congestive heart failure.

What objections are there to the routine use of digitalis? It seems to me there are very few. There is the objection that digitalis adds one more toxin to an already toxic patient, but that is rather vague. I, personally, have seen very few cases (I have seen two or three) where digitalis was given in proper dosage in pneumonia, where I think it did harm by acting as a contributing cause to a heart block of a severe degree. Such cases have been very few indeed. Over-digitalization is a very easy matter, and over-digitalization, of course, is bad for a patient and bad for the patient's heart. It is a difficult matter oftentimes to fit exactly the proper dose of digitalis to a patient. We should keep in mind the Eggleston method of estimating doses and base our orders on that.

Neither Dr. Albee nor I would have our medical feelings hurt if every case of pneumonia were digitalized if the dosage were carefully controlled, but we do have our medical feelings hurt when we see patients to whom the drug is given carelessly. (Applause.)

DR. LESTER C. MILLER, Worcester: *Mr. Chairman and Gentlemen of the Section:* I will leave it to you that following Dr. Albee and Dr. Hamilton there is very little left for me to say, so what I have to say I will confine very closely to one point which appealed to me in Dr. Albee's paper, that is, his comparison of the well-known toxic conditions in other diseases with the same toxic condition in pneumonia.

It seems to me that a study of this condition is the only way that we can arrive at any intelligent method of treament of the heart injury and the heart in pneumonia. It boils down to a few known factors, and one or two unknown factors. First, given a normal heart muscle in a not too old individual, with either of the two less toxic types of pneumonia, and the heart may almost be left to take care of itself. You are all more or less familiar with that type of pneumonia, which oftentimes surprises us with its severity of onset, but it is rapid in resolution.

Second, given the same heart in the severely toxic types of pneumonia and our use of digitalis, while indicated, is nearly always unavail-

ing because of the paralysis or the degeneration of the heart muscle. On the other hand, given an already disabled heart, in the first instance our task is to treat it as we would the same heart while undergoing some severe muscular exertion not connected with pneumonia. The same disabled heart in severely toxic types of pneumonia will undoubtedly fail faster than the normal heart. At present we are compelled to wait for the promised antitoxins which may in some measure make the problem of the heart in pneumonia simpler and less discouraging. There is some recently reported work on toxemias of the heart (and I hope we shall hear something about that later) and its reduction by the use of antitoxins. The heart toxemia is reduced by the use of antitoxins in all but one type of pneumonia. That has renewed my hope for a method of typing and antitoxin treatment of the different types of pneumonia that will be available for use almost as it is in the familiar typing of diphtheria and the use of antitoxin. (Applause.)

CHAIRMAN OHLER: Gentlemen, I notice that the first speaker on the program, Dr. F. Dennette Adams, has arrived, so we will call for his paper now. Dr. Adams of Boston.

SOME CLINICAL OBSERVATIONS ON LOBAR PNEUMONIA*

BY F. DENNETTE ADAMS, M.D.†

THE observations which I wish to report are based on the study of five hundred and twenty-two cases of lobar pneumonia above the age of sixteen which were admitted to the Pneumonia Service of the Boston City Hospital between September first, 1920 and April 19, 1922. For opportunity of observing the cases I am indebted to Dr. E. A. Locke, who was chief of the Service. The series represents only one-half of the total admissions, the remaining being broncho-pneumonia, upper respiratory infections, empyema, cases under sixteen, and an assortment of others admitted under a variety of mistaken diagnoses. It is not the object of this paper to present a statistical analysis or a treatise on the entire aspect of pneumonia, but only to point out some impressions and facts which, in the ordinary course of events, might be of value to the practitioner of medicine.

The typical case of lobar pneumonia, with definite history and signs of solidification, presents a picture not ordinarily confusing. The early case is often a problem, and an important one, important because of the frequency with which it is confounded with conditions quite remote. Beginning consolidation in the lung is often not detectable in the early stages—in some cases the affected area is never located without the aid of X-ray, and in such instances diagnosis depends chiefly on symptomatology.

The ordinary history of beginning pneumonia is so familiar as to need no comment, but there are certain features which cannot be too often emphasized. The opinion prevails that a cold, or upper respiratory infection customarily precedes the onset of lobar pneumonia. In our series this was not so, as indicated by the fact that one finds the history of a prodromal cold in only 136 or 26% of the 492 cases in which this fact was determined. Sudden onset without previous symptoms occurred in the other 74%. Sore throat was uncommon. It rarely precedes the pneumococcus case. In a pneumonia case presenting sore throat as an early symptom one should look for streptococcus as the etiological agent.

A true chill, because of the limited number of diseases in which it ordinarily occurs is helpful in establishing diagnosis. It is recorded as having occurred in this series in 42% of the cases, but one suspects this figure of being too high, because of the failure on the part of those taking histories always to distinguish between a true rigor and the chilly sensations which invariably accompany the onset of an acute fever. Of greater importance is the rarity of more than one chill. Occasionally one finds it in streptococcus pneumonia, but in the absence of reinfection, and even rarely then, more than one chill does not occur in pneumococcus pneumonia. Nor does it occur, it might be added, in association with the onset of pneumococcus empyema. It is a fairly safe working rule that a patient showing, during the early days of an acute infection, two or more true chills, has not lobar pneumonia. Much more apt is such a case to be one of generalized streptococcus infection, secondary to local sepsis, or of pyelitis with block.

In early pneumonia, the appearance of the patient is frequently more valuable than the signs in the lungs. Short, jerky respirations, with or without expiratory grunt, limitation of motion on one side of the chest, or the tendency toward upper thoracic or lower abdominal breathing, the look of anxiety, the full, bounding pulse and the abnormal pulse-respiration ratio make quite a characteristic picture. It is commonly taught that the earliest demonstrable sign of beginning pulmonary consolidation is the presence of a few crepitant râles over the affected area. Observation of this group of cases would indicate that slight dullness on light percussion, diminished breath sounds, with expiration slightly prolonged, and decreased whispered voice with rise in pitch and prolongation of its expiratory phase, are the first dependable findings.

*Read before the Annual Meeting of the Massachusetts Medical Society at Worcester, June 6, 1928.

†For record and address of author see "This Week's Issue," page 445.

And it cannot be too often emphasized that not infrequently by pushing the stethoscope high into the axilla one can detect signs of upper lobe involvement which otherwise might escape notice. Findings similar to those just described occurred in 48 of the 58 cases on which typical signs were not present on admission. Dullness with râles or pleural rub occurred in the remaining ten. Typical signs were never found in thirty-six cases.

With these figures in mind one cannot but speculate as to how often we classify as grippe or other upper respiratory infections cases of four to six days' duration, having moderate elevation of temperature, pulse and respiration, and moderate leukocytosis, which actually are mild lobar pneumonia, with areas of consolidation which have escaped detection.

In addition to the simpler respiratory infections, a variety of other conditions can be confused with lobar pneumonia. Pulmonary tuberculosis, lung abscess, pleurisy with effusion, pneumothorax, cardiac decompensation with hydrothorax or with simple congestion of the lungs, are the most common. I have twice seen pericarditis with effusion with high fever, rapid grunting respirations and compression of the left lower lobe, mistaken for pneumonia, and likewise because of type of fever and physical signs, several cases of chronic nephritis, with uremia and terminal pericarditis.

Streptococcus infection and pyelitis have already been mentioned. The mistake in pyelitis is more apt to occur in pregnancy, where upward pressure on the diaphragm can cause compression of the lower lobes sufficient to suggest early solidification. The history of chills, as previously noted, is of value in this connection.

There were two cases of deep seated cellulitis of the axilla diagnosed pneumonia. Both of these patients had pain in the side, high fever, leukocytosis, and were extremely ill. The extensive, deep seated infiltration of chest wall produced dullness and diminished breath sounds, and was responsible for the error.

Of even greater importance is the frequency with which lobar pneumonia and acute surgical conditions of the abdomen are confounded. Early right-sided pneumonia, especially if diaphragmatic pleurisy be present, is often mistaken for gall-bladder disease, or appendicitis. The latter is a particularly common mistake in children. Of one hundred and forty-five children with lobar pneumonia admitted to the Boston City Hospital between September 1, 1920, and April 19, 1922, only 45% were sent in with a diagnosis of pneumonia, and 17½% were sent into the hospital for acute appendicitis. (Adams, F. D. and Berger, B. J., *Journal A. M. A.*, 1922—79—1809).

The converse is not true;—one seldom sees acute appendicitis or other abdominal conditions diagnosed pneumonia. The reference to the abdomen of pleural pain, particularly when the diaphragm is involved, accounts for the error.

Since Cole and his co-workers (Avery, Chickering, Cole, and Dochez—Monograph No. 7, Rockefeller Institute for Medical Research, 1917) published their work on the differentiation of pneumococci, the determination of the type of infecting organism has been a routine procedure in most large hospitals. But, because of the difficulties of technique, it has never come into general use, nor will it, until a simple method of type determination is evolved. The type organism of any case must be known if specific serum therapy is to be used, but is not necessary with the polyvalent sera or antibody solutions which are now being tried. Otherwise knowledge of the type is relatively unimportant save for the fact that it is of some prognostic value.

Table 1 shows the frequency and mortality for the various organisms in this series.

TABLE 1

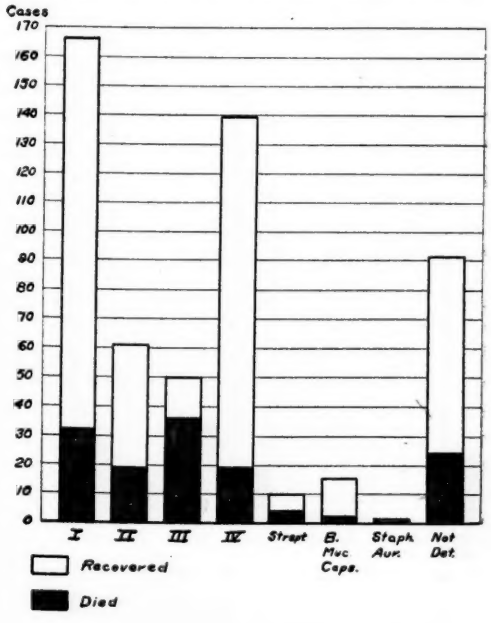
INCIDENCE OF TYPE AND MORTALITY

Type	Number	Per cent. of total	Dead	Per cent. of type	Per cent. of total deaths
I	166	31.8	32	19	23.3
II	61	11.6	19	31	13.8
III	50	9.5	36	72	26.4
IV	139	26.6	19	14	13.8
Streptococcus	10	1.9	4	40	3.0
Staphylococcus	1	.19	1	100	0.75
B. Mucosus Capsulatus	15	2.8	2	13.33	1.5
Not determined	91	17.4	24	26	17
Total	533*	101.8*	137	.....	100

*11 cases showed 2 organisms.

CHART 1

INCIDENCE OF TYPE AND MORTALITY



These figures correspond quite closely to those reported by Cecil in his study of two thousand cases at Bellevue Hospital. (Cecil, R. L.—Baldwin, H. S.—Larsen, N. P., *Archives of Internal Medicine*, 40—253—1927).

The most striking facts are the frequency of the Type I and Group IV organisms and the very high mortality of the Type III. Particularly worthy of note, especially by those who think of typing as a simple matter, is the fact that in somewhat more than one-sixth of the cases the infecting organism was not determined. This was not due to lack of effort. Usually in such cases blood cultures were negative and the patients either were not raising any sputum or a predominating organism could not be isolated by means of the mouse inoculation method. In such cases, type can sometimes be determined by culture of the small amount of fluid obtained by introducing a needle with suction into a portion of consolidated area, but it is my opinion that the end rarely justifies this procedure, and that it should not be adopted as a routine procedure for cases in which the infecting organism cannot otherwise be isolated. The blood culture often is of value as a diagnostic aid when sputum determination is unsatisfactory, but has the disadvantage of requiring more time than the mouse inoculation method.

Many workers are of the opinion that all cases of pneumonia have pneumococci in the blood stream early in the disease and that the presence of a positive or negative blood culture at this stage depends largely upon the number of organisms per unit of blood. In other words, they believe that if enough samples of blood were taken from every patient a positive culture would invariably be obtained, and that a positive blood culture obtained by the usual routine procedure indicates the presence of relatively large numbers of pneumococci in the blood, and, consequently, a severe infection.

Table 2 shows the incidence of positive blood cultures and mortality for the entire group.

With a mortality of about 61% the positive blood culture can certainly be regarded as an indication of severe infection. Of the total num-

TABLE 2

INCIDENCE OF POSITIVE BLOOD CULTURES AND MORTALITY—ALL CASES

	Number	Per cent. of total cases	Died	Per cent. Mortality
Positive blood cultures	104	20.0	63	60.6
Negative blood cultures	343	65.7	49	13.1
Contaminated or not taken	75	14.3	25	33.3
Total	522	100	137	26.2

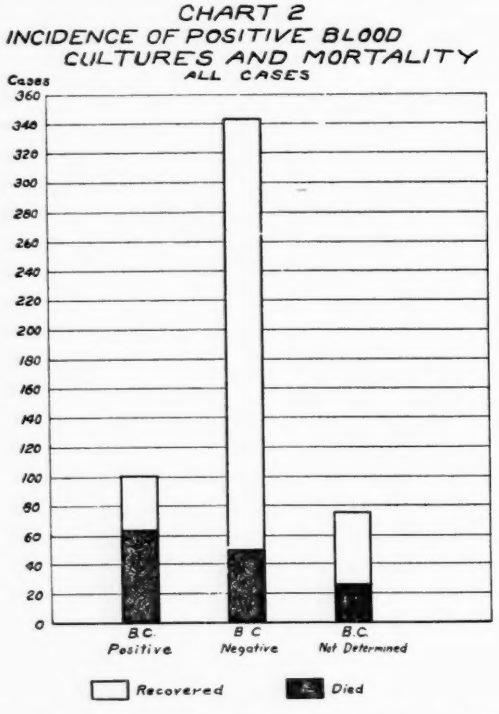
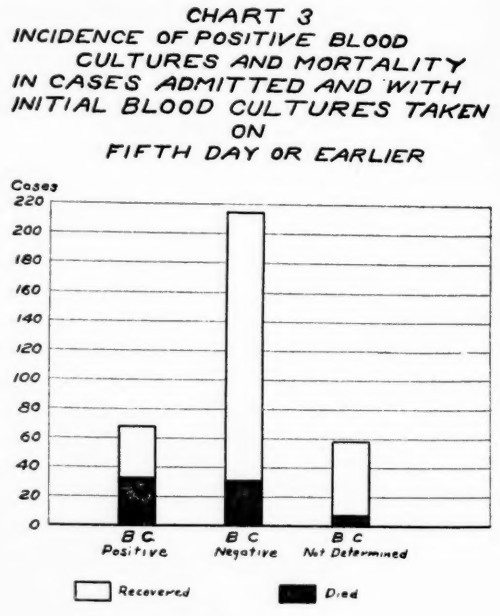


TABLE 3

INCIDENCE OF POSITIVE BLOOD CULTURES AND MORTALITY IN CASES ADMITTED AND WITH INITIAL BLOOD CULTURES TAKEN ON FIFTH DAY OR EARLIER

	Number	Per cent. of total cases	Died	Per cent. Mortality
Positive blood cultures	68	20	33	48.5
Negative blood cultures	214	62.9	31	14.3
Contaminated or not taken	58	17.1	7	12.0
Total	340	100	71	20.9



ber of cases who died, about one half had bacteria in the blood stream.

For the purpose of determining the relative severity of cases with respect to time at which cultures were found positive, the series was divided into two groups, those on whom initial cultures were taken on the fifth day or earlier, and those on whom cultures were taken after the fifth day. The results are shown in Tables 3 and 4, and Charts 3 and 4.

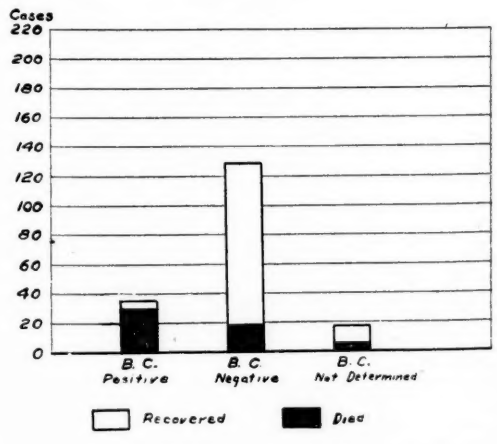
The mortality of the cases showing blood cultures positive after the fifth day is about twice that in which the positive determination was made earlier. Unfortunately, there are no figures available to show how many of the earlier group later became negative. But the figures show quite conclusively that the later a blood culture is found positive, the greater the chance of death.

TABLE 4

INCIDENCE OF POSITIVE BLOOD CULTURES AND MORTALITY IN CASES ADMITTED AND WITH INITIAL BLOOD CULTURES TAKEN AFTER THE FIFTH DAY

	Number	Per cent. of total cases	Died	Per cent. Mortality
Positive blood cultures	36	19.8	30	83.3
Negative blood cultures	129	70.8	18	13.9
Contaminated or not taken	17	9.4	5	29.0
Total	182	100	53	29.1

CHART 4
INCIDENCE OF POSITIVE BLOOD CULTURES AND MORTALITY IN CASES ADMITTED AND WITH INITIAL BLOOD CULTURES TAKEN AFTER FIFTH DAY



The leukocyte count is often of importance in diagnosis, a count above 15,000 being always in favor of pneumonia but this is not an inviolable rule. Low white counts usually occur in the mild or atypical and in a certain number of the most severe cases. More than one third of the patients who died had a leukocyte count below 14,000. In doubtful cases, a relative increase in polymorphonuclear leukocytes, 90% or over, would point toward pneumonia.

TABLE 5

LEUKOCYTE COUNTS WITH MORTALITY—ALL CASES

Count	Number of cases	Died	Per cent. Mortality
Below 8,000	34	21	61
8,000-14,000	85	29	34.1
14,000 up	400	84	21
Unknown	3	3	

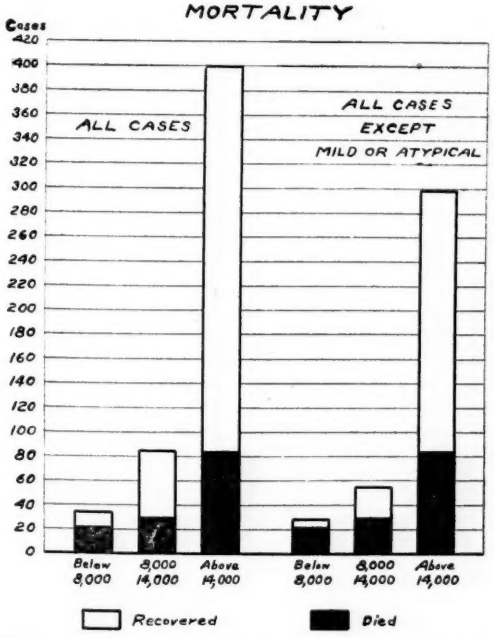
Table 5 shows the leukocyte counts for the entire series, and Table 6 shows the leukocyte counts for the series, with the mild or atypical cases eliminated.

TABLE 6

LEUKOCYTE COUNTS WITH MORTALITY
ALL CASES EXCEPT MILD OR ATYPICAL

Count	Number of cases	Died	Per cent. Mortality
Below 8,000	28	21	75
8,000-14,000	55	29	53
14,000 up	299	84	28

CHART 5 — CHART 6
LEUKOCYTE COUNTS WITH MORTALITY



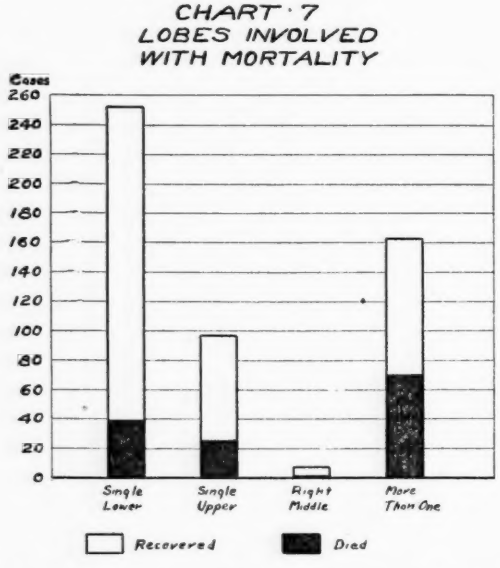
The low white count quite obviously is a poor prognostic sign. In fact, a persistently low count, in any but a mild case, is almost invariably fatal. An excessively high white count is not necessarily a grave sign. There were twenty-nine cases with a leukocyte count of 35,000 or over, and of these nine died, a mortality of 31 per cent.

Table 7 shows the frequency with which various lobes were involved. Lower lobe infection is about three times as common as the upper lobe. The latter, however, has a higher mortality rate, while involvement of two or more lobes is a decidedly more serious condition.

TABLE 7

LOBES INVOLVED, WITH MORTALITY

Lobes	Number of cases	Per cent. of total	Died	Per cent. Mortality
Single lower	252	48.3	39	15.4
Single upper	97	18.5	25	26.0
Right middle	7	1.3	1	14.3
More than one	163	31.2	70	43.0
Not known	3	.6	0	0



The relation of age to outcome was not studied statistically, there being no reason to believe that the series would show any variation from the accepted rule that, broadly speaking, beyond infancy chances for recovery decrease as age increases.

With the facts in mind which the foregoing tables have brought out, one is in a better position to give intelligent prognosis. Knowing the age of the patient, the leukocyte count, the lobes involved, the condition of blood cultures, one can estimate on the basis of probability the chances for recovery in a given case. Clinically, one recognizes as dangerous signs increasing pulse rate, irrespective of temperature, increasing respiratory rate, cyanosis, and the presence of râles elsewhere than in the pneumonic areas. No figures are available regarding the pulse—blood pressure ratio, to which, as a prognostic sign, considerable importance is often attached. In this connection it might be pointed out that a drop in temperature, sudden or gradual, is often interpreted as a favorable sign, when actually it is quite the opposite. Excluding pseudo-crisis, such a drop, unaccompanied by a corresponding fall in pulse rate, is of grave significance. Jaundice, generally considered a grave sign, occurred in thirteen cases, of which seven died. With the exception of the positive blood culture occurring late in the disease, the most disheartening single sign, I think, is a persistently low leukocyte count.

But even with all the facts at hand, one dislikes attempting a prognosis. The disease is so tricky, changes are so sudden, that in our experience one's forecast, at best, is unreliable. Sudden deaths without forewarning, sometimes associated with only a slight exertion, such as straining at stool, are not uncommon. Such deaths are apparently cardiac, yet one finds nothing at autopsy except the pneumonia. Why they occur cannot be explained. Then, too, a certain number of patients, presumably in relatively good condition, and with favorable prognosis, somewhere between the fifth and eighth days, possibly with only one lobe involved, with favorable leukocyte count, and no marked toxemia will, within a relatively few hours, become much sicker, and die. Such changes cannot be explained, much less anticipated. Occurring in my own experience, they have taught me to be very guarded in prognosis.

It has previously been shown that 385 of the patients in this series recovered. Of this number, 284, or just under 75%, were well within 11 days; 84 required 12 days or more, and in 17, day of recovery could not be determined. One hundred and sixty-two, or 42% of recoveries, were by crisis, the average day of crisis being the eighth or ninth. In only fifteen cases did crisis occur after the tenth day, and in half of these the delay was probably accounted for by extension into another lobe, or reinfection. Of the 137 who died, 105, or 77%, were dead inside of twelve days. In three-quarters of the cases, then, one can expect death or recovery within eleven days and the further beyond the twelfth day a case progresses the greater is the chance of there being some complication present.

Empyema, with thirty-five, or 6.75% of the total number of pneumonia cases, is by far the most common complication. This figure corresponds fairly closely with the 5.1 percentage reported by Cecil. (Cecil, R. L.—Baldwin, H. S.—Larsen, N. P.—*Archives Internal Medicine*, 40—253—1927.) Reinfection occurred in four cases, in three instances with the same organism, once with another; acute pericarditis in three cases; acute otitis media in five; phlebitis in four. Abscess of the lung, contrary to general impression, is rare following pneumonia. Pleural effusion with clear, sterile fluid, is relatively common. It causes no rise in temperature but may be responsible for slight continued elevation of pulse and respiration. Frequently its presence is discovered only on routine examination of the chest. The usual signs are flatness with intense bron-

chial breathing and egophony. The bronchial breathing is responsible for its being often considered unresolved pneumonia. Leukocyte count and general condition of patient distinguish such a complication from empyema. Removal of the fluid in the usual manner cures the condition. It rarely recurs.

Pneumococcus endocarditis occurred in four cases, all fatal, three diagnosed before death, and is probably of more frequent occurrence than ordinarily supposed. It should be suspected in any case characterized by the presence of prolonged, unexplained temperature after the pneumonia has presumably cleared and in which there is no evidence of empyema. A persistently positive blood culture, with myriads of organisms, is the key to the diagnosis. There are huge thrombotic masses of fibrin and bacteria on the heart valve, so that many organisms are cast off into the blood stream, and embolic manifestations are common. Changes in heart sounds may or may not be present.

Spontaneous pneumothorax, based presumably on sudden rupture of a small subpleural abscess, is not uncommon. Its onset is dramatic and alarming, but not usually fatal. Later, of course, such cases develop pyopneumothorax and, like those with empyema, require surgical attention.

Meningitis I have seen only in association with septicemia, and usually its symptoms are masked by those of the septicemia. Meningismus at the height of pneumonia is relatively common. In cases with only mild symptoms, one prefers not doing lumbar puncture.

The importance of empyema is frequently not appreciated if one may judge by the number of cases in which it is overlooked or confused with so-called unresolved pneumonia. Unresolved pneumonia is rare, and should be diagnosed only after careful deliberation. One should never be satisfied to make such a diagnosis until the patient is well; preferring while the patient is sick to continue his search for concealed pus, active tuberculosis, or pneumococcus endocarditis.

Table 8 shows the frequency of the infecting organisms.

TABLE 8

EMPYEMA WITH TYPES

Type	Number of cases	Per cent. of Empyema cases
I	12	34%
II	4	11
III	4	11
IV	11	31
Hemolytic streptococcus	2	6
Undetermined	2	6

These figures surprised me, because it has always been my impression that the Type I pneumococcus leads the field as a pus producer. I expected to find that it would account for possibly three-quarters of all the cases, and believe this would be true were we to include in this series the children and all the cases of empyema admitted with that diagnosis, and therefore not a part of this study.

It is not the province of this paper to give a differential diagnosis of empyema, but there are several points which seem worth mentioning:

In the absence of fluid sufficient to cause mechanical obstruction to respiration, a patient developing empyema will tend to get better, not worse, up to a certain point. At the time when crisis or recovery by lysis normally would occur, one frequently sees a decided change for the better, despite the presence of pus. And, following this, the patient will show slight improvement each day for perhaps a week or more, at which time increasing swings in the temperature and pulse curves will indicate beginning septic absorption. It is now generally conceded that without mechanical interference with breathing the presence of pus in the chest at this stage is not dangerous. The present-day treatment of empyema is based on these facts. Consequently, exploration is no longer considered necessary the moment the presence of pus is suspected. It is justifiable only in the streptococcus case with thin fluid, and when rapid accumulation of fluid is obstructing respiration. The cases who die early in the disease with small amounts of fluid die of septicemia or pneumonia, not of empyema. Exploratory thoracentesis as well as operation is much safer with the pneumonia a thing of the past than when the disease is at its height.

The so-called typical signs of fluid are present in empyema in slightly over half the cases. It is almost as common to find intense bronchial breathing over empyema fluid as to find sounds absent or diminished. Flatness, egophony, and absence of tactile fremitus constitute the most reliable signs.

Exploratory thoracentesis is best done at the point where occurs the greatest qualitative change in spoken voice. Change in character of the sound, particularly loss of resonance, is more important than variation in intensity.

Failure to obtain pus by exploratory puncture is often due to use of a needle of too small calibre.

If the clinical evidence is in favor of pus, countless explorations are justifiable. I have done as many as nineteen on one patient, all within an area no larger than the palm of my hand, and had my perseverance rewarded on the nineteenth attempt.

SUMMARY

1. Observations are reported in a series of 522 cases of lobar pneumonia.
2. Common factors involved in establishing early diagnosis are discussed.
3. Pneumococcus type I and Group IV are the common etiological factors.
4. Pneumococcus, type III, has the highest mortality rate.

5. A positive blood culture is a sign of severe infection and the later the blood culture is found positive the graver the outlook.

6. Save in the mild or atypical cases, a low leukocyte count is an unfavorable sign.

7. Upper lobe involvement is more dangerous than that of lower lobe, while cases with involvement of more than one lobe have the highest death rate.

8. Empyema is by far the commonest and most important complication. It is often not recognized because of the frequency with which the so-called typical signs of fluid are not present.

9. Exploration for pus in a suspected case is usually best delayed until the patient has recovered from the severe toxemia which accompanies the pneumonic infection.

CHAIRMAN OHLER: Dr. Roger I. Lee of Boston will open the discussion of Dr. Adams's paper.

DISCUSSION

DR. ROGER I. LEE: *Mr. Chairman and Gentlemen:* Dr. Adams has presented a very admirable survey of one cross-section of patients, that is to say, of the patients at the Boston City Hospital. I do not think that anybody could add anything more to such a series of cases. Some time I hope that some general practitioner is going to report a similar series of cases in general practice. I have a notion that he will find that the mortality is a great deal lower in general practice than it is in the hospital series. I think that those of us who have experience in hospital and private practice are only too well aware of that fact.

There are one or two things which come to mind in listening to Dr. Adams's paper. One of them is his really splendid courage in stating that there need be no agonizing hurry in the operation upon pneumococcus empyema. We, of course, do not want to have our empyemas neglected, but it has been, I think, the usual feeling that there is tremendous haste in the operation upon empyema. Dr. Adams has courageously brought out his own opinion, which I think is the opinion of most of us, that is that it is just as well to let the patient recover from pneumonia before he is operated upon for empyema.

I think it is the general experience that transportation is a bad thing for the case of pneumonia, and that many times it comes to us to decide as to whether the case of pneumonia should be transferred to a hospital or should be left at home. It is my strong belief that, others being relatively equal, the transportation of the average case of pneumonia to a hospital does elevate the mortality very definitely. It is, however, possible, that these cases can be well taken care of at home with their pneumonia, and then can be transferred to the hospital for operation, if necessary, for the empyema. It has always seemed to me at least, sounder practice, not to send the case of pneumonia to the hospital if he can be adequately cared for without transportation. In case of the complication of empyema, which only occurs in somewhere under ten per cent. there is an interval of a few days, a very precious few days, in which the patient usually is relatively well, and if the operative procedure can be better carried out in a hospital, then the patient can be transferred to the hospital for that operation. It is my belief that transportation at that time does the patient no harm, but that transportation at the time of his pneumonia, particularly in the first few days, is a very serious detriment to the patient and very markedly increases the mortality from pneumonia. (Applause.)

DR. WILLIAM H. ROBEY, Boston: I cannot disagree in any way with Dr. Adams's admirable paper. There are a few points I want to emphasize. Some years ago Sears and Larrabee looked over the records of lobar pneumonia at the Boston City Hospital and found that twenty per cent. of the cases had a prodromal stage. The impression which many of us have is that a good many cases of pneumonia are preceded by an acute cold. I think Dr. Adams found that in twenty-six per cent. of his cases there had been some other respiratory infection preceding the onset.

The patient's appearance is one of our greatest assets in early diagnosis; also the position which the patient takes when he is lying in bed. He is apt to lie on the affected side.

We have also paid some attention to the appearance of herpes on the lip, and also to the flush on the face, which are apt to be on the affected side in both instances. I like to ask medical students to tell which side the pneumonia is on simply from these manifestations, and more than half the time I am wrong. (Laughter.)

There is also the characteristic grunting respiration which patients have. I well remember being called once to see a boy. In that family there had been a case of acute appendicitis about four years before, with death. The attention of the physician and family was centered on the abdominal pain. As I walked along the entry to the patient's room, I heard the characteristic grunting respiration of lobar pneumonia, and felt that I was able to disregard the abdominal pain. Fortunately for me at that moment, there was an area about an inch in diameter in the right back where the pneumonia had just become apparent and I was able to point it out. Pulmonary tuberculosis rarely has the clinical appearance of acute pneumonia.

Infarction of the lung has the physical signs in the chest of acute pneumonia, but the patient rarely looks as sick and is better within twenty-four to thirty-six hours. Most cases of so-called ether pneumonia are of this type. The same thing holds of small areas of collapse, which you see in acute respiratory infections where a bronchus has been plugged and a portion of the lung has collapsed.

Dullness in early pneumonia is often a very poor sign. Sometimes there is not dullness but rather hyperresonance. You have the area of consolidation deep in the lung. There is an area of emphysema about the area of consolidation, and that for the moment will give you hyperresonance over the area of consolidation.

Another sign is the early râle, which often takes on what we like to call the consonating quality; that is, it is sharp and clear cut. It sounds as the spoken voice does, and as the respiratory murmur does. It is harsh, hard, has the sound of the consonant in contra-distinction to the soft vowel sound.

One of the most useful physical signs, in my opinion, if you suspect from the patient's appearance that you are dealing with acute pneumonia, is the spoken or whispered voice. My custom is to take the stethoscope and place it at corresponding intervals on both sides of the chest, having the patient either speak or whisper, and the very frank bronchovesicular breathing which sometimes comes through, but especially the change in the voice sound, is suggestive of the area. Mark it out with a red pencil, and in a few hours, or the next day, you will be rewarded very likely by finding that the signs of consolidation have developed.

The apex of the axilla is a very important area. I have several times seen cases where pneumonia was suspected but absolutely nothing was found in the chest until the arm was raised and the stethoscope pushed high into the axilla.

Type determination came into use at the time the Rockefeller serum was introduced, and the State Board of Massachusetts made it possible for physicians to have their cases typed.

In the clinic from which Dr. Adams has gathered cases, we were unable to type sometimes because there was no sputum. In some of those cases we tried direct punctures into the lung, but I do not like that. I think it is dangerous. In fact, I know it was in some cases, and still is.

The type determination by the State Board of Health has changed very little. In 1921, there were 486 cases typed. In 1922, 575 cases were typed, and from then on the number has gradually declined until 1927 there were 411 cases typed for the entire State, and that does not represent private practice particularly, because there are certain hospitals in the neighborhood of the State House which send all their sputum from the respiratory cases to the State Board Laboratory merely as a matter of diagnosis. Typing has its value in prognosis.

The leukocyte count is very helpful, but the low count is not always a sign of death, although it is often one of very severe infection.

I have recently seen a case of pneumonia in which the white count was persistently 8000; the temperature range as high as 104; the pulse did not rise with the temperature proportionately never exceeding 90. The case was in the care of one of the ablest practitioners I know, and after ten days and nothing had appeared in the chest, we made a diagnosis of typhoid fever, simply because there was distention, tremor, and no evidence of consolidation in the lungs. Just as the convalescence began, the right base blossomed out into a full pneumonia, and the patient began to recover.

One of the worst signs in pneumonia, apart from the low leukocyte count, is distention of the abdomen. In the army during the war, almost every soldier who had distention of the abdomen died. Those cases that can be corrected by high enema or stupes do not give us as much anxiety but a constantly increasing distention of the abdomen which can be very little relieved by our ordinary methods, whether it be enema, stupe, or small doses of pituitrin, give us very great anxiety and I consider it a very bad prognostic sign.

The true crisis also should be considered by every doctor. I have known cases where false hope was given to the family because the temperature dropped to normal. With the temperature drop must come a drop in the pulse, and above all, an appearance of well-being on the part of the patient. The doctor who is attending a case of pneumonia in a private family does not have to ask how the patient is. When he enters the front door he knows whether a true crisis has occurred. The friends of the patient will not be able to sense a false crisis, but they will almost always recognize a true one.

Patients should be kept extremely quiet during the course of the pneumonia. I do not know why it is, but I find that nurses are apparently not well instructed in this matter. If you begin to examine a patient, a nurse will immediately sit him up in bed. That should not be done. The patient should be moved as little as possible, because we know that in certain cases sudden death occurs following such exertion.

As to the use of blood cultures, when the blood stream becomes loaded with organisms, blood cultures during the end do little more than add to our prognostic information. If the blood stream is loaded at the very first, the toxemia will be profound and the outlook very poor. The worst case which I have ever seen of a blood stream loaded with organisms was a girl of twenty-five whose pneumonia lasted exactly seventeen hours. I saw her in the morning when she was comatose, the process having begun at seven o'clock the previous evening, and tried to make a blood culture. My apparatus broke, and I simply made cover-glass smears. I sent them to the laboratory, and a little later the resident called me up in great excitement and said, "Did you know that that blood is loaded with diplococci?" By that time, however, the patient was dead, and, of course, as in any of these profound cases, probably no treatment would have been of any value. (Applause.)

CHAIRMAN OHLER: The subject of the next paper is "Treatment of Empyema," by Dr. Horace Binney of Boston.

THE TREATMENT OF EMPYEMA FOLLOWING PNEUMONIA*

BY HORACE BINNEY, M.D., F.A.C.S.†

INTRODUCTORY

THE modern treatment of Empyema may be said to have begun with Bülau, who, in 1890, advocated introduction of a catheter with trocar and cannula between the ribs, connecting this with a long tube led into a bottle containing fluid —so called syphon or water-sealed drainage. In the same year Forlanini proposed gradual removal of the pus by aspiration and replacement by air, his object being to prevent the too sudden emptying of the cavity and the resulting reflex pain, coughing and dyspnoea. Both surgeons recognized that in many cases open drainage was undesirable because of the sudden pneumothorax and collapse of the lung which usually occurred. Forlanini's method received little attention until, in the late war, Morelli used it successfully in the treatment of chest wounds in the Italian army. He also advocated it in Empyema, and later in this country Davis, of Boston, called attention to it. Owing to the complicated apparatus necessary it has not met with the popularity of Bülau's method.

Even this simpler method did not come into general use until, after the disastrous results of open drainage in the earlier series of Empyema cases in the war, surgeons recognized the importance of the closed method. As a result, the mortality rate fell in a striking and convincing manner. This is all common knowledge, as is also the work of Graham and Bell who demonstrated by experiments the principle upon which the modern treatment is based, namely that pleural cavities in man are not wholly surrounded by rigid walls, but that the mediastinum, unless stiffened by adhesions or inflammatory exudate or both, is movable and therefore pressure can be easily transmitted from one pleural sac to the other. With an unstabilized mediastinum the opening of one pleural sac not only admits atmospheric pressure to the opened side but also to the unopened or sound side. The result of this is the lowering of the vital capacity of the lungs; in other words, the impairment of function of the sound lung as well as the collapsed lung on the side of the pleural opening. Also of great importance is the resulting loss of normal negative pressure upon the large veins entering the heart so that the circulation becomes impaired.

This principle was adhered to by Sauerbruch, Willy Meyer and others in the development of their cabinets for positive or negative pressure, employed in surgery of the chest, but for some reason its applicability in Empyema was not recognized until so clearly stated by Graham. Today the majority of surgeons recognize its importance in that they are agreed upon the wisdom of avoiding open drainage and consequent pneumothorax in cases either where pneumonia is present and the vital capacity low, or where the process is too early for the mediastinum to have become stabilized by adhesions or inflammatory thickening. As a result the surgical treatment of an Empyema case is no longer the grave matter or alarming spectacle of former days when, under ether, a hasty rib resection was made, a large tube plunged into the wound through a stream of pus which continued to be ejected while the wound was being closed, and in the paroxysms of coughing as the patient emerged from the anaesthetic. It is not surprising that a death on the table was not a rare event, or, if the patient recovered, that chronic empyema was too common a result.

The modern operation under local anaesthesia is a striking contrast. Now a thoracotomy between the ribs or a rib resection is done without haste, a trocar and cannula introduced through which a catheter is placed into the cavity, which is clamped off, and the pus is withdrawn by a syringe in comparatively small quantities so that cough and dyspnoea are avoided and the heart action undisturbed.

I have spoken of the importance of preliminary aspiration in the early stages of Empyema. There are of course many cases which come to the surgeon fairly late where for various reasons the presence of an empyema, complicating pneumonia, was not recognized for a considerable time. Perhaps this is seen most often in children, where the signs are more confusing, and the diagnosis of unresolved pneumonia has been made. In such cases the lung has often become adherent, if the empyema is a partial or sacculated one, or, if total, filling the whole pleural cavity, the lung is largely collapsed but the mediastinum has become stabilized by inflammatory thickening. In such cases the pus is thick or much fibrin present so that no large amount of fluid can be withdrawn by aspiration, nor is this necessary for the patient has survived the stage of great toxemia, and a rib resection and even open drainage with the development of pneumothorax are borne without any respiratory or circulatory embarrassment. Here the vital capacity is not adversely affected and may even be improved. The only question is whether closed drainage will permit adequate emptying of pus and fibrin and re-expansion of the collapsed lung. In the later or subacute cases certainly, open drainage, perhaps with the introduction of Carrel tubes for Dakin treatment, is the best method. It is a different matter, as I have already suggested, in the early cases where the patient is still toxic,

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†For record and address of author see "This Week's Issue," page 445.

the fluid sero-fibrinous or sero-purulent. In such cases preliminary aspiration has been proved to be often life saving.

In a series of one hundred cases at the Boston City Hospital reported by me in 1924 no special study was made of the results of preliminary aspiration. For the purpose of the present study I have combined with the former series the cases since that date, operated upon by me or under my direction, making a series of 126 cases. In the whole series, preliminary aspiration was carried out in 28 cases, on which we have adequate data to judge of the effect. In some the principal effect was lowering of temperature or pulse or both; in others, relief of cyanosis and dyspnoea. The amount of fluid aspirated was of course variable, always more than a simple diagnostic tapping, sometimes up to 1000 c.c. The average amount in which benefit was marked was about 250-300 c.c. in adults, a smaller amount being sufficient in children. Of the 28 cases 4 were much improved, 13 considerably improved, 11 were not improved, of which 4 died. These cases were tapped from 1 to 3 times over a period of 2 to 18 days, the average time before operation was performed being 7½ days. In one case, early in the series, large amounts of fluid were aspirated on three occasions, perhaps an example of over-zealous treatment, for the patient's condition remained unchanged, even after drainage by rib resection. He put up a hard fight for 3 weeks but finally succumbed to the virulent pneumococcus infection. I am convinced that more frequent aspiration of smaller amounts is safer, since large amounts are likely to produce reflex coughing, dyspnoea and cardiac embarrassment. Perhaps in such cases the wiser method would be aspiration with air replacement, according to Morelli's method. Our experience with preliminary aspiration is corroborated by Graham who in 1924 reported 86 cases so treated with 10 deaths. In all of the fatal cases serious complications as mastoiditis or meningitis were present. An objection often offered to preliminary aspiration is danger of spreading infection in the tissues of the chest wall. In our experience this never was serious. If Lilienthal's advice is followed, of injecting a small amount of alcohol as the needle is slowly withdrawn, infection should be avoided.

The indications for preliminary aspiration are first the presence of underlying pneumonia, second, cyanosis or dyspnoea due to pneumonia or a large amount of fluid, causing a much lowered vital capacity, and third an unduly rapid pulse from any cause. The chief contra-indication is the obvious mechanical one—namely the pus is found too thick to pass through the needle, or the presence of fibrin makes the effort futile. Here trocar-puncture and catheter drainage must not be delayed. Of course in young children or very nervous and apprehensive patients repeated aspirations may be too disturbing and a simple intercostal puncture with a small trocar and the introduction of a small catheter may be preferable. By keeping the catheter clamped off, and withdrawing a moderate amount of fluid every day or two, the same result is accomplished. In such cases however a later rib resection is often necessary and it is therefore not the procedure of choice. I have discussed this matter of preliminary aspiration at some length because I believe it to be of a good deal of importance and that it has not received sufficient attention from both surgeons and medical men.

When in a given case thick pus is definitely present, and the mediastinum stabilized, surgery is indicated and the question becomes one of choice of method. It is hardly to be expected that a group of medical men will be interested in details of surgical technique, and I therefore propose to limit myself to three points: namely first diagnostic difficulties, second, results of surgical treatment, and third, results as to occurrence of chronic empyema.

DIAGNOSTIC DIFFICULTIES

From the surgical point of view the chief difficulty of diagnosis is the matter of atypical forms of empyema: that is, the presence of small pockets of encapsulated pus, not communicating with a main large cavity, or relatively small abscesses not in contact with the chest wall such as the interlobar or so-called mediastinal empyema. Still more rare are those between lower lobe and diaphragm. An interlobar empyema by itself usually presents no great difficulty of detection, especially if adequate radiograms are available. In our series there was one case of the uncomplicated interlobar form which was diagnosed and recovered after drainage. As a complication of a simple intrapleural Empyema it occurred twice; both cases were extremely sick and died with pneumococcus septicemia. These were not diagnosed until after the main cavity had been drained. Of the mediastinal form there were two cases, one diagnosed after drainage of the main cavity, by X-ray, which showed a shadow on left side above the heart. Wide exploration and separation of adhesions behind the lung admitted a finger into a mesial pocket above the root of the lung. Drainage by a long tube was followed by recovery although the child was desperately ill.

In the second case two mediastinal pockets and several small ones between lower lobe and diaphragm were found at autopsy, the patient having died a week after drainage of a fair sized posterior axillary empyema. Needless to say they were not diagnosed. There were also two cases of apical empyema, neither of them diagnosed before operation. The first case had recovered from pneumonia 3 months before and was admitted to the hospital following an attack of hemoptysis. The diagnosis was made on the medical side of empyema which had ruptured into a bronchus, there being no evidence of Tuberculosis. As there were signs of fluid in the

posterior axillary region, corroborated by X-ray, he was tapped and 15 c.c. of pus obtained. On operation I found dense adhesions only and on wide exploration and separation of the adhesions upward my finger entered a cavity near the apex containing a small amount of pus. This was drained by a long tube. He had a slow convalescence and left the hospital with a discharging sinus. Three years later he was readmitted following an accident and his chest was found normal.

The second case of apical pocket should have been diagnosed before operation as a study of the X-ray plate revealed a dense shadow above, separated by clear lung tissue from a lower axillary shadow. After drainage of the latter, the continuance of temperature led to more careful examination of the patient and study of X-ray plates. A small area of dullness and diminished breathing was found between the spine and the upper part of the scapula; tapping brought pus and drainage was followed by recovery.

In these atypical or accessory empyemata a most careful X-ray as well as physical examination is always necessary. Often the diagnosis is in considerable doubt until thoracentesis is done, and sometimes only a wide exploration of the pleural cavity will settle the question.

RESULTS OF SURGICAL TREATMENT

1) *Mortality:* In empyema following pneumonia, what percentage of recoveries may be expected by surgical treatment? In answering this question it must be qualified in two ways: First, with regard to the method to be employed, whether closed or open drainage: second, whether the organism is a pneumococcus or a streptococcus.

As to the method, it is not my purpose to discuss at any length the relative merits of closed versus open drainage, but merely to state that as a routine method, the closed one has given better results in my experience than the open. While I have personally used the closed almost entirely, my opinion is based on the results at the Boston City Hospital. During a period of four years prior to 1920 when the open method was largely used the rate of mortality was about 20%. In the period from Jan. 1920 to Jan. 1924 there were 100 cases operated on, by me or members of my service, by a closed method as a routine, and adding to these the cases operated on by myself since 1924 there is a total series of 126 cases with a mortality of 11%.

As to the infecting organism, our experience has been the practically universal one, namely that the Streptococcus cases have a much higher mortality rate than the Pneumococcus. In the first series of 100 cases referred to there were 12 streptococcus cases with a mortality of 25%. In the pneumococcus group there were 73 cases with a mortality of 12.3%. In other words the pneumococcus cases have twice as good a chance of recovery as the streptococcus. It is fair to state, however, that many of the cases occurred during the epidemic of 1920 and 1921, when we were dealing with a more virulent form of streptococcus than in recent years.

2) *Chronic Empyema:* Coming to the last question, namely the occurrence of chronic empyema, I have no figures for comparison as to the methods employed of closed or open drainage.

A study of literature is unsatisfactory for the reason that many factors enter into the question such as age, organism present, complications, etc., and most writers have not dealt with details. In Heuer's excellent article published in 1923 he states that in 455 cases at the Johns Hopkins Hospital, 20% were chronic on admission or became so. He quotes Wilensky who found that in a series at the Mt. Sinai Hospital between the years 1903 and 1915, 23% became chronic. Doubtless most of these were treated by open drainage. Heuer states that in the four years previous to 1923 he had had no case of chronic empyema develop following closed drainage methods. Our results have been less good than Heuer's but on the whole satisfactory. There were but 6 cases of chronic empyema in 126 cases. These were treated surgically, in various ways, with five recoveries, and one death. The fatality occurred from meningitis following a decortication. This gives an incidence of chronic empyema of 4.7%.

There are several reasons why one would expect a greater frequency of chronic empyema following streptococcus than pneumococcus cases. The amount of pus, and therefore the compression of the lung are often greater, the patient is more prostrated and less able to begin breathing exercises early, so that expansion is more delayed, and the wound is more apt to break down, causing an air leakage and pneumothorax. Another reason is the far greater incidence of bronchial fistula in streptococcus cases. Naturally with fistula present, neither Dakin's fluid nor breathing exercises can be used—conditions which greatly favor the development of chronic empyema. These are conclusions supported by our experience. While data as to bacteriology are not complete, it is evident that a much greater proportion of streptococcus, than of pneumococcus cases become chronic.

Our results confirm us in the belief that preliminary aspiration is often of great value, and that, in accordance with the experience of many others, as a routine procedure the closed method, combined with irrigation with Dakin's fluid, gives a lower mortality rate than is obtained by open drainage methods, and also a lower incidence of chronic Empyema.

CHAIRMAN OHLER: The formal discussion of Dr. Binney's paper will be opened by Dr. Edwin A. Locke of Boston.

Discussion

Dr. Edwin A. Locke, Boston: Mr. Chairman, Ladies and Gentlemen: Dr. Binney has given us a very concise and authoritative statement of the treatment of empyema occurring with pneumonia, as well as very extraordinary statistics of his results surgically. I have no opinion to express regarding the method of surgical treatment of empyema, nor have I any right to any authoritative opinion. It seemed to me, however, that it is more a question of resourcefulness, judgment and conscientiousness on the part of the surgeon than of the particular method of surgical treatment. It has been my privilege to see the majority, at least, of these cases on which Dr. Binney bases his experience, as they were drawn largely from the pneumonia service and the fourth medical service at the City Hospital. I believe that it has been the skill and conscientious care given the cases by Dr. Binney which has given him these truly remarkable results, and not necessarily the closed method of treatment.

His figure of eleven per cent. mortality in his operated cases is perhaps made more striking by contrast with figures which I have for the last twelve years' experience at the City Hospital in the treatment of empyema. Very briefly, those twelve years are divided into three periods. The first period is from 1915 to 1919, when, as Dr. Binney has stated, the open method was used entirely. The mortality during that period was 16 per cent. During the period of the pneumonia service, namely, from 1919 to 1923, the final combined mortality figures were 22.7 per cent. For the period 1923 to 1927 the case fatality rate was 16.6 per cent., and for the entire period of twelve years, 21.5 per cent. These figures, of course, are not quite comparable to Dr. Binney's, inasmuch as some of these cases for various reasons never came to operation. I think, however, that these figures over a period of twelve years are sufficient to show the splendid quality of the work which Dr. Binney has done.

Dr. Binney and Dr. Adams have both spoken at some length regarding the diagnosis of empyema. We are all conscious of the great difficulties which sometimes attend the making of an accurate diagnosis, especially in some of the severe cases with a large amount of fluid. There are one or two practical points that I should like to make, even though they are perhaps very well known. In the first place, it seems to me of the utmost importance to watch the temperature. The pyrexia in empyema is often very slight, there being only a slight afternoon rise to 99-100°. I have seen one or two cases without temperature. Leukocytosis is, on the whole, a more reliable sign of infection of the pleura, I think, than is the temperature curve. I have never, so far as I can recall, seen a case of empyema that failed to show a leukocytosis. The most elusive types, as Dr. Binney has stated, are those so-called encapsulated forms which occur not infrequently. In most instances, physical signs, if carefully looked for, are fairly characteristic. I have never been able to diagnose the rare cases of encapsulated empyema in the mediastinum or between the diaphragm and the lung, and in these cases the X-ray seldom helps us. Finally, the necessity in many instances of needling the chest for diagnosis is imperative, and I do not believe that any man is so expert in the examination and interpretation of signs in the chest that he can always feel sure of the presence or absence of empyema.

I must take issue with Dr. Robey in what he has said with regard to the dangers of lung puncture. On the pneumonia service we had 71 cases where we deliberately did lung puncture for diagnosis, without the slightest evidence of injury to the patient. It is not a dangerous procedure in our experience, although I must confess seldom necessary. I am speaking, of course, of a consolidated lung and not a normal one. This is comforting because it very often must happen that we puncture a lung when making a diagnostic thoracentesis of the chest.

Dr. Binney has spoken of the usefulness of preliminary aspiration in cases of empyema. This is a procedure which is of considerable value in many instances, although obviously not to be recommended as a routine procedure. I agree with him that the amounts removed should be relatively small, probably from three to five hundred c.c. If the technique is properly carried out, this procedure can be done without any great inconvenience to the patient.

The indications for this form of treatment prior to operation are found in two types of cases. First, in cases entering the hospital with a very large amount of pus and who are in a very critical condition, preliminary parecentesis should be done and perhaps repeated. This type of case we quite frequently see in large hospitals. In former years when such cases were immediately treated by open drainage the patient commonly died on the table. The second type is the streptococcus empyema which almost always occurs with bronchopneumonia. In these cases it is often imperative to do a preliminary puncture or several at intervals of one to several days before it is safe to attempt permanent drainage in any form. In other words, in these so-called metastatic forms of empyema operation must be deferred until the patient is over his pneumonia and his general condition stabilized.

There is one very important difference between the time of occurrence of streptococcus and pneumococcus empyema which has been hinted at once or twice this morning, but not as emphatically stated as I feel it should be. Streptococcus empyema usually occurs, during the course of the pneumonia, most commonly with bronchopneumonia and especially in the forms which we saw with the influenza epidemic. The fact that it develops coincidently with the pneumonia makes it practically impossible to do an early oper-

ation. On the other hand, the pneumococcus empyema occurs as a sequella of lobar pneumonia when the patient is more or less stabilized so far as the toxemia is concerned.

The type of organism is of some importance as regards both the treatment and the prognosis. In our entire series treated on the pneumonia service 67 per cent. were due to the pneumococcus, 27 per cent. to the streptococcus, and 6 per cent. to the staphylococcus. The rather large incidence of streptococus infection is probably due to the fact that during the first year we had a great many of the severe cases of influenza on the service. Seventy-five per cent. of the pneumococcus empyemas were of type 1 infection, illustrating the well-known fact which has been brought out by Dr. Cecil and the other workers in the Rockefeller Hospital, that the type 1 pneumococcus is the one which most commonly leads to complications in the nature of focal infections. The streptococcus is a more virulent organism than the pneumococcus. As Dr. Binney has stated, in streptococcus empyema very large amounts of pus may accumulate in an incredibly short time.

When the results of treatment are stated as showing a case fatality ratio of 20 to 22 per cent., we must remember first that we are dealing with a very virulent form of infection, whether it be due to the pneumococcus, streptococcus or staphylococcus. With all of these there is considerable chance of other focal or general infection.

Dr. Binney has given the mortality figures for his twelve streptococcus cases as 25 per cent. Our much larger series showed 30 per cent. Dr. Binney's 73 pneumococcus cases showed a mortality of 12.3 per cent., ours 15.7 per cent. The mortality rate in our staphylococcus cases was 40 per cent.

In closing, I want to emphasize in just a word the importance of what one might well call the medical treatment of empyema. So often as one observes empyema treated in the hospital, it seems to be regarded purely as a mechanical procedure. It has always seemed to me that the medical treatment, so-called—in other words, the program which one carries out to combat, to help nature rather to combat an infection—is just as important in the treatment of empyema as the operation itself.

CHAIRMAN OHLER: Unfortunately, Dr. Hunt of Worcester is unable to be present, so the formal discussion of Dr. Binney's paper will be closed by Dr. Royal Watkins of Worcester.

DR. ROYAL P. WATKINS, Worcester: Dr. Hunt found at the last moment that he could not come, and I am here as a pinch-hitter for Dr. Hunt, without much time to prepare. He had made a few notes of things that he would like to offer, and I will try to cover those points somewhat.

Dr. Binney's paper lays down correct principles of the surgical treatment of empyema, which none of us is inclined to dispute. Of course there are always variations in some details of the carrying out of any principles of treatment which are governed more or less by the personal preference of the surgeon who is doing it.

I should like to emphasize what Dr. Locke has emphasized somewhat, viz., the importance of the needle in the diagnosis. It not only determines whether the pus is there and locates it for us, but it gives us an opportunity to get some pus for a culture, and he has pointed out the obvious advantage of having that culture previous to the time of operation.

The value of the local anesthetic in these operations is, I think, also undisputed. Whenever it is possible to do either the closed drainage or the rib resection with the local anesthetic, it certainly is the best way to do it. There are, however, occasionally cases in which it cannot be done, notably in very young children. My experience in empyema dates back quite a number of years, to the time when I was an interne in Bellevue Hospital in 1892. For a good many years we did not use local anesthesia, of course. We operated on a great many empyemas. We used ether. The results were not so alarming to me as they seem to have been to some other people, and I, therefore, do not feel so much upset in any particular case if I have to use a general anesthetic. I do know, however, that the local anesthetic is much preferable.

Dr. McLaughlin, one of our internes at the Worcester City Hospital, has reviewed a series of 103 cases, all operated cases and of all types, done at the City Hospital since 1920. There were 103 cases of all types, and the deaths numbered 11, giving a mortality rate of 10.7. In 87 cases a rib resection was done. In 9 of these 87 cases a second operation was necessary, and in 3, a third operation, making about 14 per cent. requiring reoperation. The intercostal drainage was used in 13 cases, a closed method. In most of these the flap or valve method was used in the drainage to prevent influx of air and allow the exit of the pus. Intercostal drainage was done in 13 cases, 4 of which, or 31 per cent., had to have a rib resection later.

The bacteriology is rather interesting. The pneumococcus was the cause of 51 of the cases. That is a little less than 50 per cent. The mortality in the pneumococcus cases was 7.84 per cent. The streptococcus cases of all types were 20, with a mortality of 20 per cent. I have here the various types of the streptococcus, but perhaps I will leave those out.

The staphylococcus cases were 14, with a mortality of four per cent. Mixed staphylococcus and streptococcus, 4 cases, of which none died. Sterile cultures occurred in 3, with 33 1/3 per cent. mortality. In 10, there is no record of culture.

There were 7 chronic cases, of which one died. This was a three-year old child who had primary inter-costal drainage through a No. 18 catheter. This was followed two and a half months later by a rib resection when the patient came back to the hospital, after having been discharged. She died of a complicating pyemia.

I should like to sum up in my own fashion, regardless of the notes given me by Dr. Hunt. I do not know whether he would concur in this or not. It seems to me that the treatment of empyema can be summed up in about this way: Of course we have to have our diagnosis; we have to know when to operate, when is the proper time to operate, and select the operation according to those things. Some cases must be aspirated or provided with some sort of intermittent drainage to tide them over an early dyspnea or cyanosis, but all the cases must in the end have adequate drainage. Opinions differ as to the method of securing this. My own observation and experience have convinced me that I can get the best results by a rib resection if the section removed is not less than two inches long and is placed at the lowest point in the pus cavity. (Applause.)

CHAIRMAN OHLER: The title of the next and last paper of the morning is not quite as printed in the program. The title is "The Recent Advances in the Specific Treatment of Pneumonia."

Gentlemen, it is a pleasure and an honor to be able to introduce to this section Dr. Russell L. Cecil, formerly of the Rockefeller Institute, now in the Bellevue Hospital, New York. (Applause.)

RECENT ADVANCES IN THE SPECIFIC TREATMENT OF PNEUMONIA*

BY RUSSELL L. CECIL, M.D.†

Mr. Chairman, Ladies and Gentlemen:

DR. ADAMS has pointed out to you the significance of septicemia in pneumonia. All students of pneumonia are agreed that the patient very ill with pneumonia is nearly always septic. For that reason, immunologists have directed their attention in this direction, and from work on monkeys, we are convinced that if we could sterilize the blood of all of our pneumonic patients we could cut the mortality rate from the standard 30 per cent. of big city hospitals to somewhere around 8 or 10 per cent. I do not think one can ever expect to get a specific for pneumonia that will give a hundred per cent. of recoveries.

Pneumonia is a very different disease from diphtheria and scarlet fever and these other infections that yield brilliantly to specific remedies. Pneumonia, in the first place, is a disease that attacks mostly middle aged and elderly people, and those who are alcoholic or have some other systemic disease. We do not have the same healthy young organisms to work on that we have when dealing with diphtheria or scarlet fever. It is remarkable to see how a healthy monkey responds to serum when he has been infected with type 1 pneumonia; but then these monkeys are never alcoholic! We are dealing with healthy young organisms and all we have to do is to fight the pneumococcus.

We have been interested in the specific treatment of pneumonia for seven or eight years at the Bellevue Hospital. We started with the type 1 serum, and then were anxious to try a serum for the other types, working along on monkeys in the laboratory and on patients in the wards. Some work was started several years ago with Huntoon's derivative of serum which consisted of immune bodies in a water solution. Some interesting results were obtained with it, but the reactions were often severe, with chills and fever, and with one or two fatalities, so we were rather afraid of it, and furthermore it was not as potent as we wanted.

About three years ago Dr. Lloyd Felton offered to prepare a new extract of serum for us, a derivative obtained by precipitating out the globulins from immune serum. I will not go into that process, but this Felton's serum that I am going to talk to you about is a polyvalent product which is made by immunizing horses against types 1, 2 and 3 pneumococcus. It contains no immune bodies against type 4 pneumococcus, but it has been used on type 4 pneumonias as a sort of check on the other types to see if it had any value in this type also. The horses, after they have been immunized, are bled and the serum is prepared in the usual manner. Then the immune bodies are precipitated out with distilled water and redissolved in a concentrated form. The final product is slightly opalescent and ten to twenty times as strong as the original serum. This, of course, is a great advantage in giving the serum because instead of having to use some complicated apparatus to give 100 or 150 c.c. of fluid, we take a Luer syringe and put from 10 to 20 c.c. of serum directly into the vein.

Felton's serum has a nitrogen content about like that of normal serum and a specific gravity of 10.32. It has the consistency of ordinary horse serum, but of course in using so much less, the total amount of foreign protein injected is much less than one would get with ordinary serum. For that reason the serum complications such as serum disease and anaphylaxis are not so marked.

As I said before, this product is potent. We have now developed a method of standardizing antipneumococcus serum in units, and the unit is the amount of serum that will protect a mouse

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†For record and address of author see "This Week's Issue," page 445.

against a million fatal doses of pneumococcus culture.

The polyvalent Felton serum contains on an average of about 2000 units against type 1, about 2000 against type 2, and a much smaller amount, 200 to 400 units against type 3. So while it is very potent against types 1 and 2, it has comparatively little potency against type 3, and as a result probably of this low potency against type 3, it has little or no effect in controlling type 3 infections either in monkeys or in man.

The experiments on animals are very satisfactory, particularly in type 1. One can give monkeys perfectly classic lobar pneumonia with pneumococcus type 1 injected intratracheally, injecting many times the fatal dose. If after the disease has progressed for a day or two, intravenous treatment with Felton's serum is begun, invariably the blood is sterilized and the temperature begins to come down, the monkey gets well, while the control monkeys die. It is a very satisfactory method of testing out the value of these specific agents, one that we use a great deal in research work.

We have tried to develop in Bellevue a reliable method of determining the therapeutic value of these products. We take every case as soon as the diagnosis of lobar pneumonia has been made, and that case is given a number. About 500 lobar pneumonias a year are admitted to the twelve medical wards at Bellevue. The even numbers get Felton's serum as soon as the diagnosis of lobar pneumonia is made. The odd numbers get no serum, but in other respects are treated in the same way. It is very important to get the serum treatment started as soon as possible.

The typing is one of the most important parts of our work from the scientific point, because it enables us to know what we are doing in each particular case. In private practice, typing is not so essential, but it is a nice thing to have. In research work it is absolutely essential to have accurate typing, and even with the best typing in the best laboratories, with specially trained people to do it, there is probably an error of 10 to 15 per cent. in the reports. The only way to overcome that is to have repeated specimens examined and check up with blood cultures, cultures from pleural exudates, urine precipitin tests, and so forth.

Tests for hypersensitiveness to horse serum are made before the serum treatment is started. We generally do an intradermal test, but we always do an ophthalmic test, because we find the ophthalmic test is more sensitive as to horse sensitivity than the skin test. The patient is asked if he has had asthma or hay fever or ever received serum of any kind before. The ophthalmic test will generally indicate if the patient is very sensitive. If the patient is sensitive, we proceed with great caution, giving very small doses to desensitize the patient. If the patient's history is negative and the ophthalmic test is negative, we proceed at once to give 5 c.c. of Felton's serum intravenously, watching the patient to see if any symptoms of anaphylaxis develop. After the 5 c.c. has been given, we give larger doses, and try to get in about 80 to 100 c.c. of Felton's serum on the first day of the patient's stay in the hospital. This means a great deal more than 100 c.c. of the old-fashioned serum or standard serum, as it is 10 to 20 times as strong. On subsequent days, the number of injections is governed by the condition of the patient. If there is a rapid drop in temperature and improvement in the patient, the dosage on the second day would be much smaller. The average dose is 20 to 25 c.c. intravenously.

Occasional reactions have been noted. One can't give any kind of serum or serum derivative without getting occasional reactions. The chills that sometimes follow the intravenous injections are caused by a foreign protein. We discard the lots that give chills, or send it back for repurification. Most of the lots now come in perfect condition and give no reactions of a foreign protein nature.

The other reactions are the hyper-sensitive reactions in patients who are allergic to horse serum. We have had a few anaphylactic reactions. A syringe of adrenalin is always at hand so that if the patient begins to get a little cyanosed, the adrenalin can be administered at once. We have not had any fatalities in the 414 cases treated with Felton's serum. There has been a smaller incidence of serum sickness than with the original standard type 1 serum. Serum sickness was noted in 10 to 12 per cent. of cases and seemed to depend a good deal on the amount of serum which the patient received. Patients who get a large amount of serum are very likely to develop serum sickness a week to two weeks after the injection.

Remember that the immunologists are trying to sterilize the blood in pneumonia. These serums that we are working with are not antitoxic serums. No toxin has been isolated from the pneumococcus; there is no antitoxin against the pneumococcus. These specific agents that are being tried out in pneumonia are antibacterial. Their function is to increase the agglutinins, precipitins, and opsonins in the blood, and to hasten phagocytosis of the pneumococci.

Figure 1* shows a typical chart of a type 1 pneumonia. The crisis appeared on the seventh day. The blood culture taken every day was sterile, and at the time of crisis the patient began to develop immune bodies in his blood. That is what happens in all types of pneumonia with recovery. The patient, whose blood has been free from any evidence of immune substances, about the time of the crisis, or shortly after, begins to develop these immune bodies. If the blood culture has been positive, the immune

*In the following charts the upper solid line represents the temperature, the lower solid line the daily blood cultures. The dotted line indicates the amount of protective substance in the circulating blood.

bodies never appear until the bacteria disappear from the blood.

The object in giving serum to any type of pneumonia is to help Nature establish a balance of immune bodies in the blood in order that it

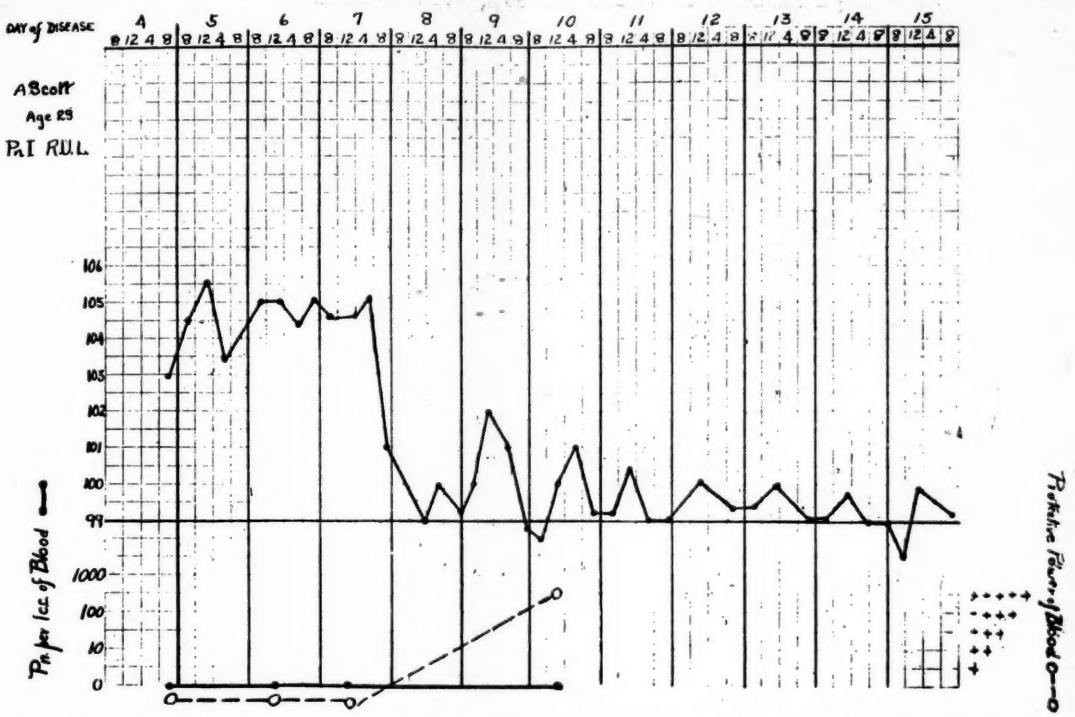


FIGURE 1. Type 1 pneumonia. Crisis on 7th day. Immune bodies appear in circulating blood just after crisis.

will be impossible for pneumococci to circulate there; we have learned that with rare exceptions, pneumococci and immune bodies do not circulate in the blood at the same time.

The patient with pneumonia needs a great deal of help; he needs more help relatively speaking than the patient with diphtheria or scarlet fever, because he has a very large infected area. The diphtheria patient has small patches in the throat and the scarlet fever patient has a red throat infected with streptococcus. Compare these small localized infections with a man who has three lobes solidified with pus and innumerable pneumococci. You can see how much more of a problem we have in trying to overcome such an infection than we have in dealing with infections such as scarlet fever and diptheria.

Table I indicates what Dr. Adams has already brought out. The death rate in patients with positive blood cultures is much higher than in patients with sterile blood cultures. These patients had almost daily blood cultures. We found that where the blood culture was positive, seventy-eight per cent. died; where the blood remained sterile, only ten per cent. died.

Type of Pneumococcus	Cases Studied	Cases with positive blood culture		Cases with sterile blood culture	
		Cases	Died	Cases	Died
I	39	11	6	28	3
II	22	14	13	8	1
III	11	4	3	7	1
IV	35	8	7	27	2
TOTAL	107	37	29	70	7
			(78.3%)		(10.%)

Table 1.- Death rate in patients with positive blood cultures compared with death rate of patients with sterile blood cultures.

Figure 2 shows a type 1 pneumonia treated on the third day with two injections (5 c.c. and 20 c.c.) of Felton's serum. There was a drop in temperature on the morning of the fourth day. Two extra injections were given to prevent any recurrence.

Figure 3 shows a type 1 pneumonia, treated on the first day, the ideal day to treat any case of pneumonia with serum. The temperature, which was 104.5°, began to come down immediately. After four injections of serum on the first day, the temperature on the second day was 100°. Several extra injections were given to prevent a relapse. This patient shows a rapid development of immune bodies. Ordinarily we would not expect them to appear until about the seventh or eighth day. Here is a very large balance of immune substances in the blood on the second day. This accounts for the early crisis, to some extent, at least.

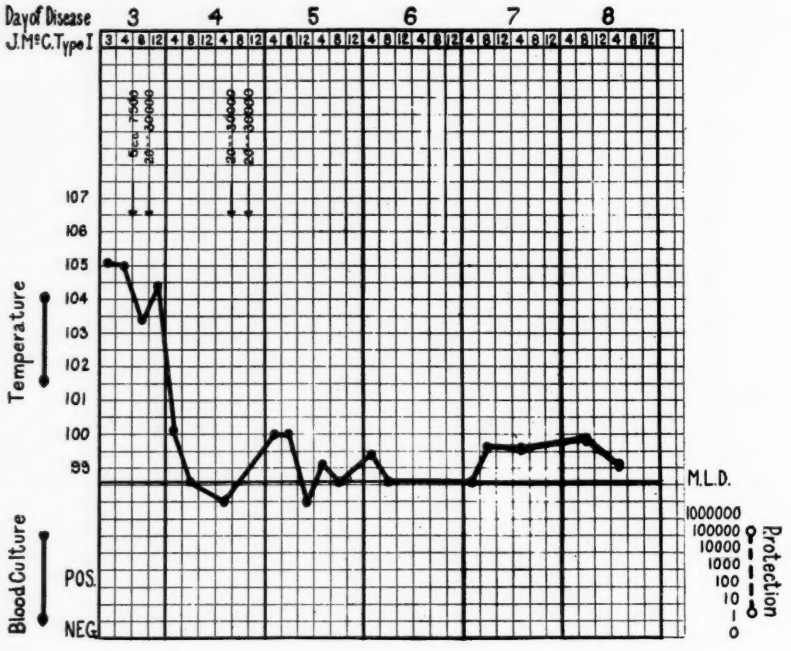


FIGURE 2. Type 1 pneumonia treated with Felton's serum.

Figure 4 is a type 1 patient treated on the third day, with a drop in temperature after one injection of Felton's serum. Several extra injections were given on the fourth day. This is a particularly striking case, because the patient came in with a positive blood culture. As the pneumococci disappeared from the blood, immune bodies appeared in the blood, the tem-

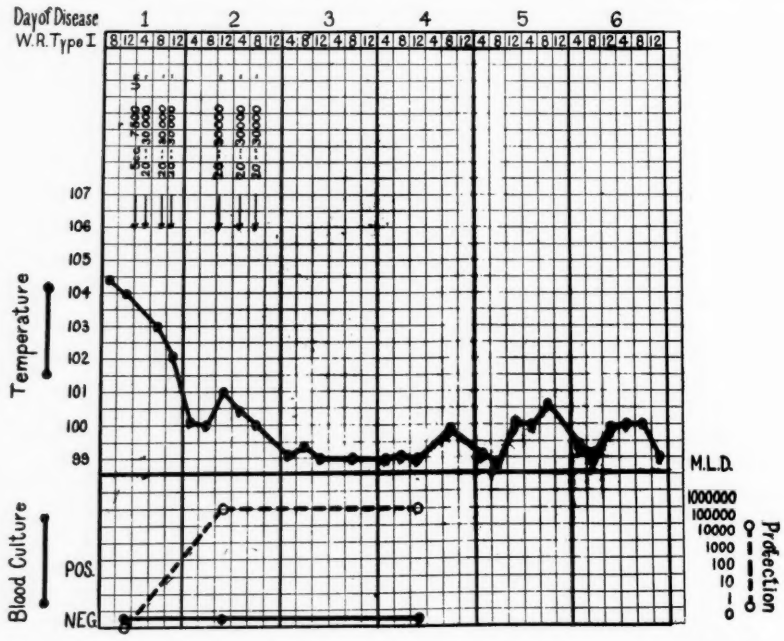


FIGURE 3. Type 1 pneumonia treated with Felton's serum on first day of disease. Blood cultures sterile. Protective substance appearing in blood on second day.

perature came down and the crisis was on the end of the fourth day.

Figure 5 shows another nice type 1 case, also septic, treated with Felton's serum, with the well clinically. I think it must be due to the unresolved exudate in the lung, because they are perfectly well, except for a little fever for four or five days.

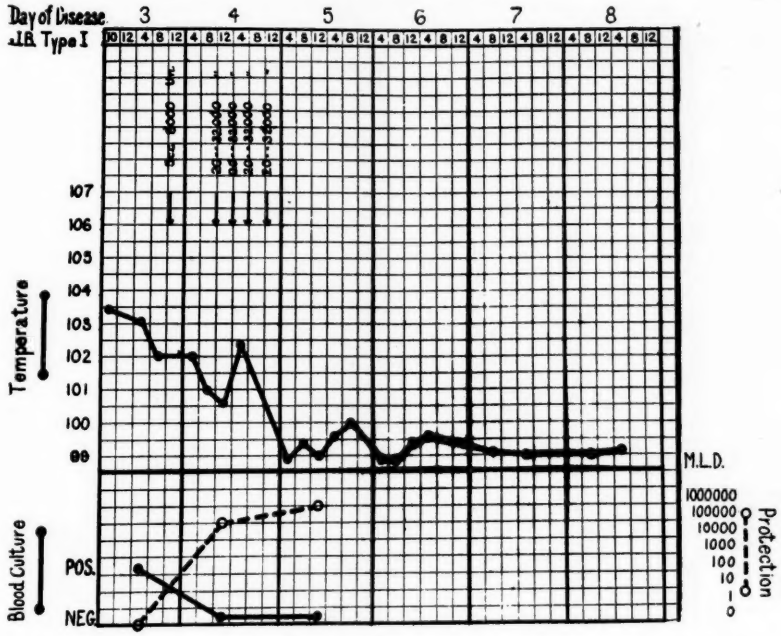


FIGURE 4. Type 1 pneumonia treated with Felton's serum. Blood sterilized after one injection of serum. Protective substance in blood after first injection.

bacteria disappearing, immunity appearing, and the temperature coming down on the third day.

Figure 6 shows a septic type 1 patient with a temperature drop after two injections of serum. Quite a number of our cases have gone on running a little fever after they are apparently

Figure 7 shows another type 1 chart very much like the preceding one, with the crisis on the fourth day, sepsis disappearing and immune bodies appearing on the fourth day.

The problem is essentially the same for type 2 as for type 1 pneumonia, except that for type 2,

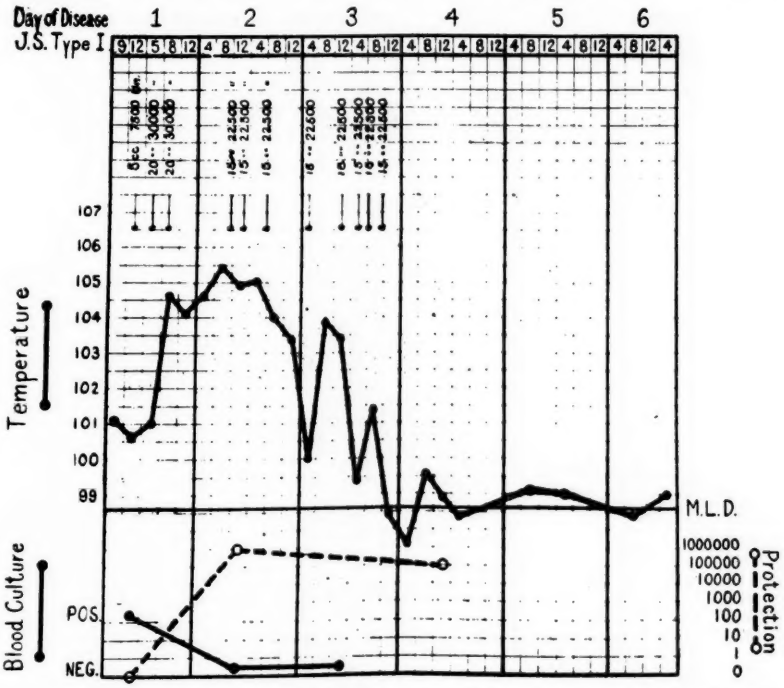


FIGURE 5. Type 1 pneumonia treated with Felton's serum. Blood culture sterile and protective substance appearing in blood after three injections of serum.

where the death rate is almost twice as high as it is for type 1 pneumonia, we need a still more potent serum, and that has been a very hard thing to develop. With the development of potent serums, the treatment of type 2 pneumonia

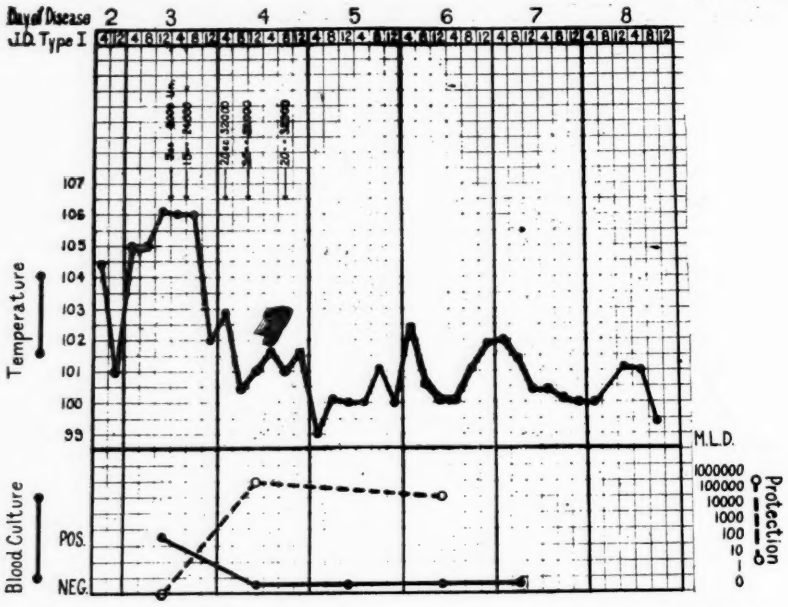


FIGURE 6. Septic Type 1 pneumonia treated with Felton's serum. Blood sterilized and protective substance present after three injections of serum.

becomes quite feasible and at Bellevue we believe we are making a dent in the mortality rate of type 2 pneumonia, which, runs a death rate

the fifth day there was a kickup, but altogether it was a rather satisfactory clinical effect.

Figure 9 shows another type 2 case. There was a very spectacular drop on the second day. This patient was treated with one large dose of serum. One does not often see such a quick drop in type 2 pneumonia.

Figure 10 is the chart of a nice type 2 case,

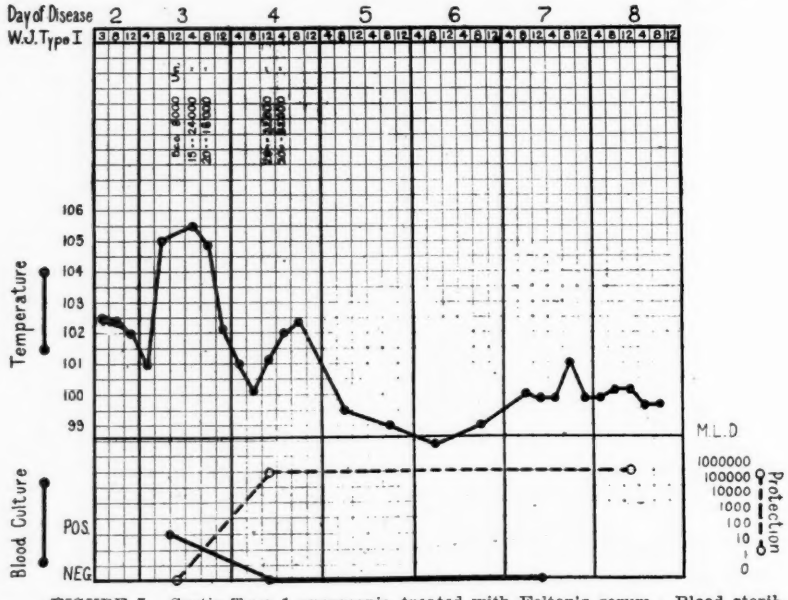


FIGURE 7. Septic Type 1 pneumonia treated with Felton's serum. Blood sterilized and protective substance present after three injections of serum.

higher than the type 3 cases, around fifty per cent. in untreated cases.

Figure 8 shows a type 2 chart of a patient who came in on the third day. We treated it rather intensively on the third day. On the fourth day, the temperature began to drop. On

treated on the third day, with immune bodies appearing immediately after the introduction of serum, just as in the treated type 1 cases.

Figure 11 is a type 2 pneumonia with a positive blood culture, treated on the third day with three injections of serum. There was a drop in

temperature on the fourth day. The blood culture became sterile. Immune bodies appeared in the blood, and crisis occurred on the morning of the fifth day.

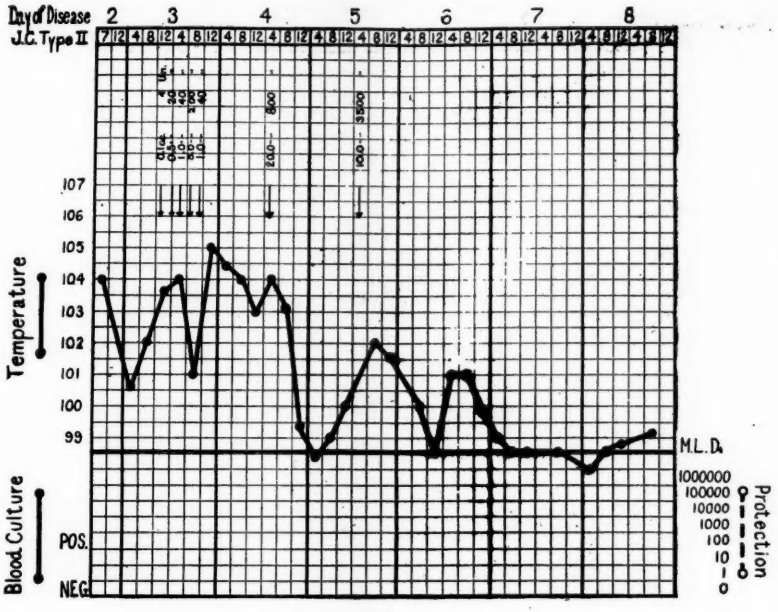


FIGURE 8. Type 2 pneumonia treated with Felton's serum.

Figure 12 shows a composite temperature chart that we recently prepared from twenty-one treated cases and twenty-one untreated cases, all of which recovered, both the treated and the treated cases. This bore out our clinical impression that we were shortening the disease considerably by the early administration of Felton's serum. We have not prepared a composite chart for type 2 cases. I do not believe it would be as striking as the type 1 chart. During the last two years we have treated 414 cases of lobar pneumonia of all four types with Felton's serum,

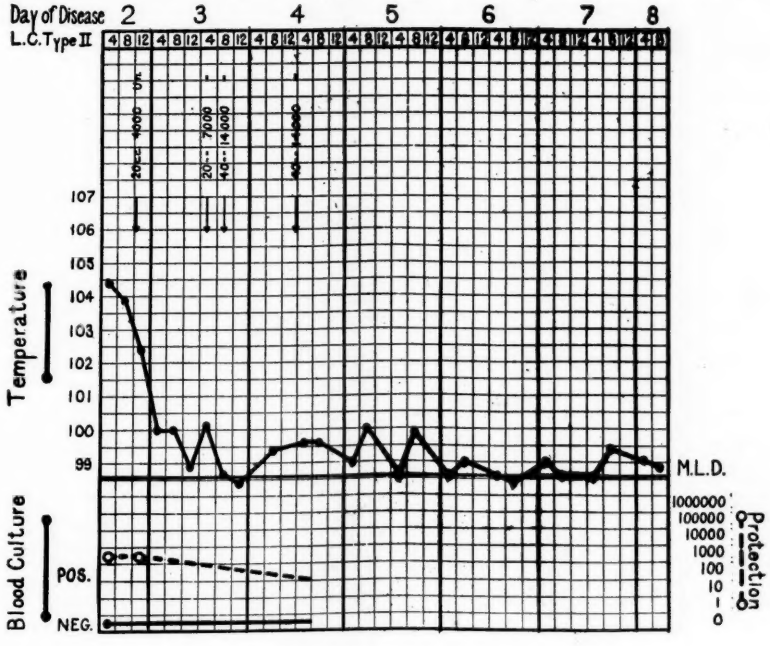


FIGURE 9. Type 2 pneumonia treated with Felton's serum.

untreated. These cases were all type 1 infections. These cases were not selected with any regard to the clinical effect; they were selected because they were early cases. In the treated cases, the composite temperature came down about two days earlier than it did in the untreated and with that, 407 controls that received no serum. In Table 2 you will see that the death rate in untreated type 1 pneumonia is pretty high—thirty-two per cent. In type 2 it runs up to fifty-two per cent. in ninety-five cases. In type 3, the death rate ought to be higher, as

type 3 has always run a high death rate. For two years before this our type 3 death rate was above forty per cent. Turning now to the treated cases, type 1 shows the best results. In 144 cases of type 1 pneumonia the death rate was only eighteen per cent., compared with thirty-two per cent. in the controls. There is no question of doubt in my mind that Felton's serum is a very valuable specific in the treatment of type 1 pneumonia. In type 2 the results are not so striking, thirty-nine per cent. in the treated, compared with fifty-two per cent. in the untreated. In type 3 the death rate was actually higher in the treated cases than in the untreated. In the type 4 group, there was some difference in favor of the treated cases. In fact, we believe

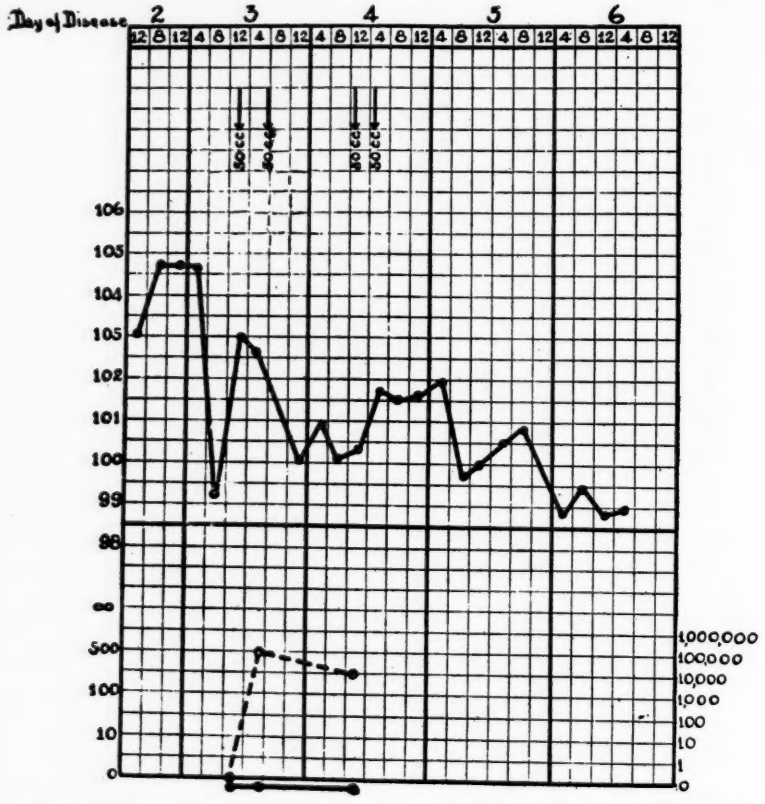


FIGURE 10. Type 2 pneumonia treated with Felton's serum. Protective substance appeared promptly at the institution of serum treatment.

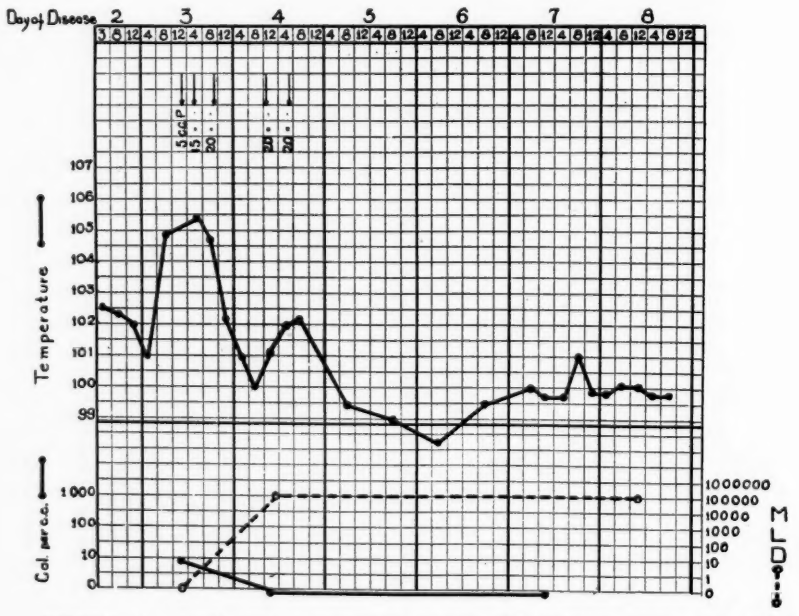


FIGURE 11. Septic Type 2 pneumonia treated with Felton's serum. The blood sterilized and immune bodies present in the blood after serum treatment.

that we can do even better in type 4 pneumonia, and this year Dr. Park is planning to make a special type 4 serum. He has been working on the sub-groups of type 4, and has found that there are seven dominant type 4 strains. He is going to take these dominant subtypes and immunize horses and make a special type 4 serum, so that when we are dealing with a type 4 infection we can switch off the polyvalent serum and give our patient a special type 4 serum. Type 4 pneumonia is generally of a milder type than type 2 or 3, and ought to yield to treatment almost as well as type 1, if we get the right strains with which to work.

I always dislike to show statistics from Bellevue Hospital, because even after they have been treated the figures look pretty bad. Twenty-eight per cent. sounds like a very high death rate, and yet it is a good deal lower than thirty-eight per cent. when you think that that means ten lives out of every hundred patients.

Unfortunately, the patients come to Bellevue very late; they do not get in early. If we take out the early cases (Table 3), the three-day cases, we get a death rate in type 1 pneumonia of only eleven per cent. I think we could reduce the death rate of type 1 pneumonia to less than ten per cent. if we could treat them all during the first three days. If you could treat them during the first forty-eight hours, it would be still lower. The mortality rate for the whole group is twenty-three percent. for the early treated cases, as compared with twenty-eight per cent. of the early controls.

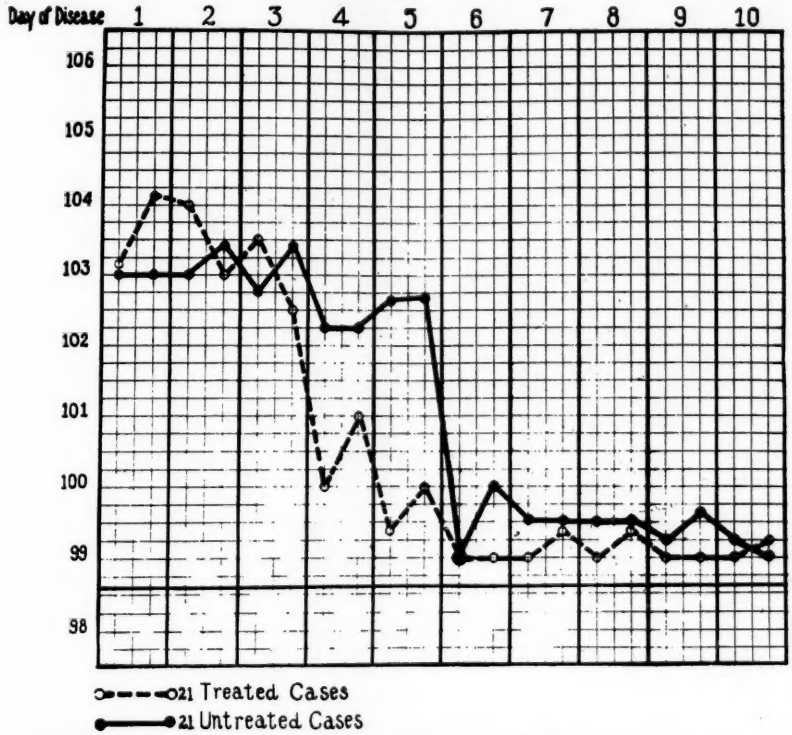


FIGURE 12. Composite temperature chart of 21 treated Type 1 pneumonias compared with the composite temperature chart of 21 untreated cases.

	TREATED			UNTREATED		
Type	Cases	Died	% Mort.	Cases	Died	% Mort.
I	144	27	18.6	132	43	32.6
II	107	42	39.2	95	50	52.6
III	32	12	37.5	52	14	26.9
IV	131	39	29.8	128	51	39.8
	414	120	28.9	407	158	38.8

Table 2.- Death rate in 414 cases of lobar pneumonia treated with Felton's serum, compared with 407 untreated control cases.

We hope to continue this work for another two years at least, going on very much as we have been going in the past two years. It takes a long time to get enough statistics to really convince yourself and to convince other people. After all, though, the thing that really gives us confidence in Felton's product is the clinical effect that we get, and the effect we get on monkeys. Statistics, of course, are always hard to work with, and as I say, it takes a great many cases to make figures convincing.

To summarize we may say that in Felton's serum we have a potent product, a polyvalent product; we have a stable product; we have a practical product, something that is easy to administer and which does not give severe reactions.

We hope to have within the next year a polyvalent type 4 serum which we can use in type 4 pneumonia and thus have a specific agent for three out of the four forms of lobar pneumonia. (Applause.)

sometimes ten million fatal doses of pneumococcus.

We have also realized that antipneumococcic serum has in it some substance antagonistic to its protective power so that if we use very large doses of the anti-pneumococcic serum, we sometimes do not get as good an effect as we do if we use smaller doses.

Recently there has been some work done in the Harvard Medical School by Mr. Goodner, who has very kindly allowed me to present some of his findings, which are going to be of great value not only in the study of pneumococcus infections but also in the testing of antipneumococcic serum. Mr. Goodner, by injecting virulent pneumococci into the skin of rabbits, produces a lesion which is progressive, and which in many cases resembles in a very striking way the processes in lobar pneumonia. This lesion goes on and increases both in size and in intensity. Bacteremia is produced and nearly always the animals die with all the symptoms of

	TREATED			UNTREATED		
Type	Cases	Died	% Mort.	Cases	Died	% Mort.
I	54	6	11.1	51	13	25.5
II	50	18	36.0	40	15	37.4
III	11	3	27.3	18	2	11.1
IV	51	12	23.6	38	12	31.6
	166	39	23.5	147	42	28.6

Table 3.- Death rate in cases of lobar pneumonia treated during the first three days of the disease, compared with control cases admitted during the first three days of the disease.

CHAIRMAN OHLER: The formal discussion of Dr. Cecil's paper will be opened by Dr. Benjamin White of Boston, the Director of the State Antitoxin Laboratory. (Applause.)

DISCUSSION

DR. BENJAMIN WHITE, Boston: *Ladies and Gentlemen*—Dr. Cecil has shown admirable perseverance in the way he has continued his studies on serum treatment of pneumonia, and I think you will agree that his perseverance has been rewarded. I have had the feeling that his perseverance has been due to the fact that he has looked at pneumonia not only from the standpoint of a clinician but also from the standpoint of a bacteriologist and immunologist. All of us who have been interested in pneumococcus immunity have realized that if we inject pneumococci, dead pneumococci, into experimental animals, those animals respond by developing agglutinins, precipitins, protective substances, and other antibodies in their blood. We also know that this serum in certain amounts will protect experimental animals against a million or lobar pneumonia only produced in the dermal way.

With this method he has been testing out therapeutic serums, and he finds that if you inject the ordinary therapeutic serum into an animal with a lesion of this type, if you give enough serum and if you give it soon enough, you prevent the death of every animal, but there is a very interesting time relation which he has observed in regard to the amount of serum which is necessary to save an animal at different periods. There is apparently an exact mathematical relation between the dose of the serum to be used on any given day, and if we can interpret his results and apply them to human treatment, it would seem that in late cases of pneumonia we are not successful in influencing the infection, because we cannot give a large enough dose of serum. He will give a dose of serum to his animals which would represent an injection of four or five hundred cubic centimeters of serum into man, which, of course, is obviously impossible.

He shows also that when this serum is injected there is an immediate sterilization of the blood in these animals, and if the dose is large enough, the blood remains sterile and the animal then recovers by crisis at the time of the injection.

We have also realized that the serum which has been produced has not been as strong as we would wish in antibodies, and I think great improvements are coming about in this regard. Some unpublished work of Avery's, done at the Rockefeller Institute, seems to indicate that we can produce serums of much higher antibody content than we have ever produced before, and we are now instituting this method in our laboratory in the immunization of horses.

Then with the work of Felton, in freeing these protective antibodies from the other serum constituents in antipneumococcic serum and by concentrating the antibody, we feel confident about the future of the serum treatment, antibody treatment, of pneumonias.

We have been so convinced by Dr. Cecil's work that at the laboratory in Boston we are now immunizing our horses against both type 1 and type 2 pneumococcus. Our chemists are working on the concentration process, and next year we will be able to distribute the Felton antibody solution. This will not be given out for general distribution, but will be given to clinicians who will use it and who will give us reports of their observations. (Applause.)

Dr. Frederick T. Lord, Boston: *Mr. Chairman*—The problem of specific treatment of pneumonia has been before us now for a number of years. It rests upon a firm foundation. In experiments on the smaller laboratory animals it can readily be demonstrated that homologous immune serum will prevent and cure an otherwise fatal pneumococcus infection. Antipneumococcus serum and Felton's antibody will also cure an otherwise fatal pneumococcus pneumonia in monkeys and for this important contribution we are indebted to Cecil and his associates, who have thus established the value of specific therapy for monkeys and encouraged its further trial in man.

Laboratory investigations have made it clear that recovery or death in pneumonia is chiefly due to a balance of opposing forces. There is, on the one hand, the production by the host of antibodies which can be demonstrated in the blood serum at about the time of the crisis by protection tests on mice. Baldwin and Cecil and Park and Cooper have shown that by the use of Felton's antibody solution it is possible to establish a positive balance of protective substances in the patient's blood. In the course of the infection on the other hand there is produced by the invading pneumococci an apparently non-toxic substance capable of neutralizing or destroying antibodies and enhancing the virulence of the organism. This inhibiting substance is produced in larger amount as the infection proceeds and, hence, early use of specific therapy may be expected to lead to better results.

I do not propose to go into details regarding the earlier results with serum, but I would like to call attention to a collected series of 553 cases of Type 1 pneumococcus pneumonia treated with unrefined Type I antipneumococcic serum with a mortality of 15.9%, while in 301 untreated Type I cases the mortality was 24.9%.

Our experience with specific therapy in Type I pneumococcus pneumonia embraces a series of 81 cases treated intravenously with antiserum. Seventy-one were treated with unrefined serum and 10 with Felton's antibody solution, with 17 deaths, a mortality of 20.9%. During the period covered by the serum treated group, 80 patients with Type I pneumococcus pneumonia, not so treated, have been observed with 19 deaths, a mortality of 23.7%. Though there is a suggestion of benefit in the slight lowering of mortality in the treated series, the death rate in the two groups is so nearly the same as to warrant no definite conclusion on the series as a whole as to the value of the method.

Cecil has referred to the discouraging aspect of statistical studies in estimating the results of specific therapy. In one of his earlier reports (*Arch. Int. Med.*, Sept. 15, 1927) there is a series of 352 cases of Type I pneumococcus pneumonia not serum treated with 73 deaths, a mortality of 20.7%, or practically the same as in our treated group. In 144 Type I treated cases in his present report there is a mortality of 18.6%. While his control series shows a lowering of the mortality from 32.6% to 18.6% in favor of the method, on comparison with his previous series there is a reduction from 20.7% to 18.6%. It seems probable that the expected mortality in Type I pneumonia is about 20%.

When, however, the treated group in our series is divided into those given specific therapy early and those to whom it was given late there is a more striking difference in the figures. Of 28 cases treated within the first three days only 2 died, a mortality of 7.14%, and of 53 treated after the third day 15 died, a mortality of 28.3%. Considered alone, this number of cases is too small to be conclusive, but in addition to the 28 cases in our experience and the 54 just reported by Cecil, there are 12 by Locke, 13 by Christian, 56 by Cecil (treated with Huntoon's antibody), and 29 by Bullowa, making in all 192 Type I pneumonia cases treated with antiserum or its derivatives within the first three days with 21 deaths, a mortality of 10.9%. The question has been raised whether among early admissions to hospitals the death rate may not be lower than the average, but from our own and the experience of others, this can be answered in the negative and the number of cases and the low mortality in this combined treated series indicate curative value in specific therapy given early in the disease.

I may say then that from our own and the ex-

perience of others, there seems to be no doubt of the value of this method of treatment in Type I pneumococcus pneumonia when the anti-serum or antibody is given within the first three days of the illness and I may say further that there seems relatively little benefit from such therapy after this interval. Early adequate dosage is most important for success. It is important to appreciate, as Cecil has pointed out, that Felton's antibody permits early use and sufficient dosage. Prior to the introduction of this antibody solution it was generally conceded that it was unjustifiable to treat all patients with pneumonia with unrefined Type I antipneumococcic serum and thus subject two-thirds of the patients to troublesome reactions without possibility of benefit. Felton's antibody, however, has greater potency, is polyvalent, and much less likely to cause reactions than unrefined serum. A dosage of 5-10 c.c. intravenously obviates the necessity of complicated apparatus. Its use before the type of pneumococcus infection is determined saves at least 24 to 48 hours, a precious interval in the treatment of pneumonia. It is not only of value in Type I, but probably also in Type II pneumonia. Its use does not eliminate the desirability of typing the infection, however, as a guide to continuance or cessation of specific treatment. In spite of the absence of any severe reactions after the use of Felton's antibody, I would emphasize the importance of observing the customary precautions against the use of alien serum in hypersensitive individuals by an inquiry regarding a history of asthma, hay fever or a previous injection of horse serum. It should be appreciated that a large group of children have now been sensitized to horse serum by the preventive inoculation with toxin-antitoxin mixture against diphtheria. Perhaps a quarter of the individuals thus protected against diphtheria have been sensitized to horse serum and will continue to be sensitive for years.

We are now confronted with the important question whether or not we should use Felton's antibody solution in the treatment of lobar pneumonia. In the answer to this question we must consider not only the advantage to the patient which seems established when specific treatment is given early in the disease, but also the possibility of danger from the use of alien serum. The experience thus far seems to indicate that under the conditions in which Felton's antibody has been used it is harmless and if due precautions are exercised there need, I think, be no hesitation in its application to individuals who are not hypersensitive to horse serum.

I would like to say in conclusion that much credit is due to Cecil for his contributions to a better understanding of the pneumonia problem and for his carefully controlled experiments in specific therapy.

CHAIRMAN OHLER: According to the program, we are due at the Main Hall of the Chamber of Commerce Building shortly to attend the annual meeting. I think we will all agree that we have had a most interesting meeting. I am sorry that the program did not work out so that there was time for general discussion, but at any rate I think we have all been well repaid.

The following officers were elected to serve during the ensuing year:

Chairman, Lester C. Miller, Worcester.

Secretary, Roger W. Schofield, Worcester.

The meeting adjourned at twelve-ten o'clock.

POSTERIOR MEDIAN DISCISSION OF THE CERVIX

Indications For This Operation, And A New Technique For Its Performance

BY SAMUEL R. MEAKER, M.D., F.A.C.S.*

THE os externum of the nulliparous cervix is normally a small aperture. Its smallness causes no trouble, even in "pinhole" cases, so long as the endocervical secretions are normal. If, however, the secretions become infected or viscid, the situation is complicated by the factor of faulty drainage.

Thus it happens that chronic endocervicitis in nulliparous patients is often persistent and obstinate. Free drainage is the first requisite in the treatment of any localized infection; failing this, all other treatment is likely to be ineffectual.

Thus, also, it may happen that viscid secretions are retained. In such event they soon become inspissated, and ultimately form a thick and tenacious mucous plug. This fills, and sometimes distends, the cervical canal; the secretions grow more and more abnormal; and so a vicious circle is established which cannot be broken so long as there remains an obstacle to free drainage. The presenting symptom in such cases is usually sterility.

There are, therefore, two main conditions the cure of which, in nulliparous women, may require some surgical procedure that permanently enlarges the os externum of the cervix. One of these is chronic endocervicitis, and the other is viscosity of the endocervical mucus. They are common conditions, occurring both singly and in combination.

Many operations have been devised to overcome stenosis of the os externum. The one most commonly performed is simple dilatation; that, however, is wholly inadequate in the conditions under discussion, since the effect of stretching on muscular tissue is very transitory. Bilateral splitting was introduced by Simpson, developed by Sims, and improved by Pozzi; it has the objectionable feature that, like an obstetrical laceration, it often produces ectropion of the endocervical mucosa. Posterior median discission with plastic reconstruction has been done in various ways and is exemplified by the well-known operation of Dudley; the results of Dudley's operation are usually excellent, but occasionally the drainage provided is not adequate.

The operation here described has numerous advantages. It is easily and quickly performed, and entails only two or three days' hospitalization. The primary object of the operation, free drainage, is attained in the fullest degree. The endocervix is made readily accessible for local treatment, and yet at the same time is not exposed to the vaginal environment. A posterior enlargement of the os externum facilitates the natural mechanism of insemination. The plastic result causes no obstetrical difficulty.

This simple operation, followed, if necessary, by appropriate after-treatment, will often obviate the need of more radical surgical procedures such as the operations of Schröder or of Sturmdorf.

TECHNIQUE OF OPERATION

The perinaeum is retracted with a weighted speculum or a ribbon-retractor. The anterior lip of the cervix is seized in the mid-line with a volsella forceps, which draws the cervix down within easy reach. The cervical canal is thoroughly dilated. Two traction-sutures are now introduced through the posterior lip, one on either side of the mid-line; they are cut long, and each is clipped with a haemostat. When these sutures have been placed, the volsella is removed from the anterior lip.

The next step is the discission of the cervix. One blade of a stout pair of straight scissors is introduced within the dilated cervical canal, and the posterior lip is divided in the mid-line for one-half or two-thirds of the distance up to the os internum. (Fig. 1.)

If the discission is done exactly in the mid-line, there will be little bleeding. It is usually advisable, however, to make firm pressure against the cut edges for a minute or two and thus to secure a dry field for the subsequent plastic work.

With a sharp knife the vaginal covering of the cervix is now undercut about half-an-inch deep all along each side of the split. It must be mobilized sufficiently to make a flap that will cover the raw muscular surfaces. (Fig. 2.)

For suture-material No. 1 chromic catgut is used. While each stitch is being introduced, the mobilized vaginal flap should be drawn toward the operator and held taut. All sutures are placed before any are tied.

The needle enters the vaginal surface of the posterior lip of the cervix, and emerges from the muscle near the cut edge of endocervical mucosa. It then pierces the vaginal flap near its free edge from within outward. Returning, it pierces the flap from without inward, enters the muscle, and emerges from the vaginal surface of the posterior lip. (Fig. 3.)

As a rule, five such sutures are required—one in the mid-line at the top of the split, and two on each side. (Fig. 4.) Occasionally one or two superficial stitches will be needed in addition to secure the nicest coaptation of the cut edges. At the completion of the operation the two traction-sutures are removed.

*For record and address of author see "This Week's Issue," page 445.

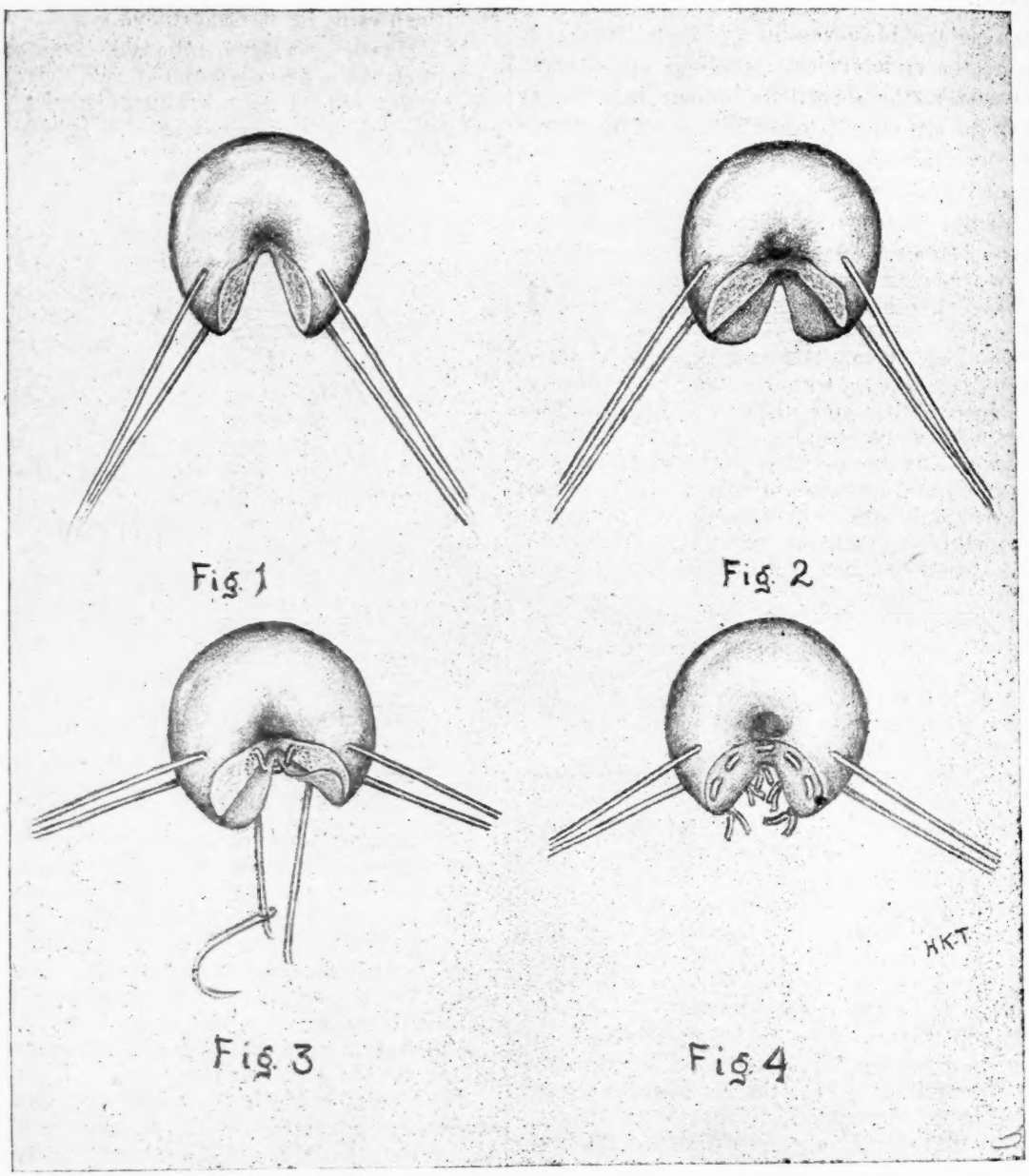
ILLUSTRATIVE CASES

CASE 1

Mrs. D. E. consulted me for sterility at the age of 25. She had been married to a first husband from 17 to 20, and to her second husband since the age of 22. In neither marriage had there been a pregnancy.

CASE 2

Miss M. P. consulted me for leucorrhoea, present some six months. The discharge was thick and purulent, and profuse enough to necessitate the constant use of a protective napkin. For several months she had been under more or less continuous treatment, having tried without benefit all varieties of douches, tampons, suppositories, and other local measures. The performance of the Sturmdorf operation was under consideration.



A complete sterility-study was carried out, dealing with the husband as well as with the wife. It revealed only one definite abnormality: the cervical canal was filled by a thick and tenacious plug of inspissated mucus, and the endocervical mucosa was obviously unhealthy, bleeding at the slightest touch. Postcoital examination showed in the cervix a few spermatozoa, dead and entangled in viscid mucus, while in a condom-specimen the spermatozoa were found to be numerous and active.

Posterior median discission of the cervix was done. The patient missed her third period, and was uneventfully delivered less than eleven months after operation.

Examination showed the endocervix to be the source of her discharge. The infection was apparently non-specific, for at no time, either before I saw her or afterward, was the gonococcus demonstrated, nor was there any urethritis or other characteristic evidence of gonorrhoea.

Posterior median discission of the cervix was done, and long hot douches of plain water were ordered as after-treatment. Within six weeks the discharge had stopped and the cervix appeared healthy. The patient was kept under observation for about a year, during which she remained free from all trouble.

LARYNGEAL TUBERCULOSIS

A Plea For Its Early Diagnosis

BY SAMUEL CLINE, M.D.*

THE early diagnosis of laryngeal tuberculosis is of paramount importance if the process is to be arrested, or a cure effected. The writer's experience, covering the past eight years in a sanatorium for tubercular patients, has brought him in contact with many cases of laryngeal tuberculosis in which, in not a few instances, the process was unrecognized in its early stages and treatment instituted too late. Many patients whose first symptoms are related to the larynx and who are totally unaware of any process in the chest present themselves.

The student has been taught that laryngeal involvement is an end stage of advanced pulmonary tuberculosis and, therefore, that the condition is seen only in sanatoria or in bedridden cases. In a large measure this is true, but the degree of laryngeal involvement does not always depend upon, nor correspond to, the extent of the pulmonary process. Generally speaking, there is such an interdependence; however, the instances are numerous in which advanced throat lesions are found in conjunction with small active or even arrested foci in the lungs. Conversely, we find patients in whom widespread pulmonary involvement runs its course without manifest laryngeal disease.

Not infrequently we find moderately advanced lesions of laryngeal tuberculosis in routine examinations of patients with mild throat symptoms. It is the lack of recognition of the possibility of laryngeal tuberculosis in these ambulatory and apparently well patients that has led to delay in the administration of proper treatment and in some instances unwarranted operations which have ended disastrously.

SYMPTOMATOLOGY

In early laryngeal tuberculosis one encounters voice changes ranging from slight huskiness to hoarseness. Fatigue of the voice is a prominent symptom, so that, after use, the voice is noticeably weaker and lacks its clear resonance. Other sensations may be present in the throat such as a scratching feeling, tickling and excessive dryness. Dysphagia, when present, is not an early symptom and implies an advanced process.

DIAGNOSIS

The examination by the use of the mirror may be difficult if the patient's reflexes are uncontrollable. However, by the aid of cocaine applications, the pharynx may be put at rest and the larynx easily examined. The technique is simple and a conscientious practitioner should give his patient the benefit of an early laryngeal examination. With the continued use of the mirror one can become fairly adept at recognizing the normal larynx and thus be in a position to note any variations from the normal. In the sanatorium are seen many patients in late stages of laryngeal tuberculosis who, previous to admission, had never had the benefit of a laryngeal examination.

Anemia of the mucous membrane has generally been considered an early sign, but I have usually failed to note it except in the advanced states. A general or localized congestion of the larynx, such as may be consistent with a catarrhal laryngitis, ordinarily clears up within a few weeks. However, if the trouble persists, one must bear in mind the possibility that the laryngeal condition is more serious and requires further investigation. The earliest and most characteristic manifestation of laryngeal tuberculosis is a swelling or infiltration of one or more parts, most commonly in the interarytenoid sulcus. The infiltration may persist indefinitely without any change and may present a smooth surface with a grayish color. Occasionally, an acute swelling of one of the arytenoid cartilages marks the first appearance of the disease.

Next in frequency to the involvement of the arytenoids or interarytenoid space is that of the vocal cords. The circumscribed swelling is most common in the posterior part of the cord, with rapid extension to the remaining portion. Ulcerations, when present, are very suggestive of tuberculosis and are broad and shallow with gray bases and ill-defined outlines.

TREATMENT

The disease is by no means as irremediable as it has been commonly thought, and with early recognition one can offer some hope to the patient. Sanatorial care is essential, as the condition of the lung must be treated coincidentally with that of the larynx. Complete rest of the voice, so that even whispering is forbidden, is helpful in many cases in arresting the disease. In certain selected patients, the use of the galvano cautery applied to the individual lesions has offered the greatest help. Topical applications of lactic acid or trichloracetic acid are useful in other cases. Direct sunlight thrown into the larynx by aid of a suitable mirror has been extensively used in sanatoria in the West. Alcohol injection, and more recently resection of the superior laryngeal nerve, have aided in quieting the larynx and alleviating pain.

The following recent cases will serve to illustrate the above points:

CASE 1. Grace L., 19 years, salesgirl, had been

*For record and address of author see "This Week's Issue," page 445.

hoarse for the past six months, with dryness of the throat and slight pain referable to the left side of the throat. A diagnosis of chronic tonsillitis was made at one of our hospitals and tonsillectomy was advised. There was no examination of the larynx. Tonsillectomy was performed in April, 1926. Her laryngeal symptoms became worse and one month later she was admitted to the Boston Sanatorium with an advanced chest condition and laryngeal complications. She died at home in October, 1926, six months after the tonsillectomy.

CASE 2. John B., 28 years, policeman, came into the Out-Patient Department with the complaint of huskiness of the voice for the past two months. Examination showed some interarytenoid infiltration with a little thickening of the right arytenoid. There were no symptoms suggesting pulmonary tuberculosis and the patient stated he felt fine. Examination of the lungs disclosed some involvement at the right apex.

He was sent to a Sanatorium and his last report (after three months' stay) stated that his throat had become normal and the pulmonary process quiescent.

CASE 3. Marion L., 36 years, school teacher, for the past two months had suffered with dryness of the throat and fatigue of the voice so that, at the end of the school session, huskiness was pronounced. She considered her general health good, but had a very slight cough without sputum.

Her larynx showed moderate swelling of the right arytenoid extending to the posterior part of the right cord. Careful examination of the chest showed a suspicious process in the right apex, which was confirmed by X-ray. She is now undergoing sanatorium care and is showing improvement as to her laryngeal condition.

SUMMARY

1. Early diagnosis of laryngeal tuberculosis is imperative if treatment is to be successful.
2. Neglect of laryngeal examinations leads to the overlooking of early cases, which soon become advanced.
3. The first symptoms of tuberculosis in many patients are referred to the larynx and they may be totally unaware of any process in the lungs.
4. Any acute laryngeal condition which persists, without improvement, more than a few weeks, requires further investigation.
5. Practice with the mirror and, if necessary, cocaine applications will enable one to become familiar with the normal larynx and thus to be in a position to recognize deviations from the normal.
6. Many patients have been subjected to ill advised operations as a result of symptoms caused by an underlying laryngeal condition.

RESULTS OF THE MASSACHUSETTS CANCER CAMPAIGN

APRIL 23-27, 1928

BY FRANKLIN G. BALCH, M.D., F.A.C.S., GEORGE H. BIGELOW, M.D., AND ROBERT B. GREENOUGH, M.D., F.A.C.S.

UNDER legislation passed two years ago there have been developed cancer clinics in fifteen cities outside of Boston, and the State cancer hospital at Pondville has been opened. In addition there is, of course, the service at the Huntington Memorial Hospital and other special cancer service in hospitals and dispensaries both in and out of Boston developed in many instances over many years and without any reference to any special State program. All this gives a more readily available service for cancer than has been developed over any similar area and of as high a general level of quality. This being the case, it was felt that the time had come to inform both the public and the profession of the resources and of their proper utilization. This was the Cancer Campaign which was organized jointly by the Committee on Cancer of the Massachusetts Medical Society, the Massachusetts Branch of the American Society for the Control of Cancer, and the State Department of Public Health. The authors constituted a Campaign Committee representing these three organizations.

The plans as outlined by the Campaign Committee called for:

(1) A mass meeting in each city in which there was a State-aided clinic.

(2) A request addressed to each of the eighteen District Medical Societies that a special meeting to consider cancer control be held during that week.

(3) Special clinics for physicians to be conducted in connection with the regularly established cancer clinics.

(4) The co-öperation of the larger industrial, mercantile, and public service concerns by the distribution of pay envelope slips and bill enclosures; by publishing in their house organ an article on cancer control from material to be supplied by the committee; and by arranging for speakers to address their employees.

(5) Extension of the scope of the speakers' bureau through an enlarged staff of speakers which included a number of eminent medical men from outside the State.

(6) An information booth centrally located where literature and information would be available.

(7) Extensive use of the press and the radio.

The local medical cancer committees and cancer education committees handled the programs in their own cities.

THE PRESS

Preliminary notices were sent out on March 30, April 10, and April 17. A form for an editorial was also sent to each paper. Clipping returns show that many of them carried either

*For records and addresses of authors see "This Week's Issue," page 445.

this editorial or comment based upon its content.

At this stage an experienced press agent was placed in charge of all news service. Each day's program was announced on a State-wide basis a day in advance of its occurrence. Clipping returns accounted for 2,838 column-inches of space. This represents, of course, but a small portion of the actual amount of space given before, during, and after the campaign.

PRINTED MATTER

A new pamphlet "Cancer? Be Quick!" was prepared by the Department of Public Health, telling a story of progress in the Cancer Control Program inaugurated by the Department in 1926. This was distributed at all meetings, in various ways by local committees, and was sent out with mail by the Department during the week of the campaign. Approximately 30,127 copies of these were distributed.

A flyer entitled "Help Fight Cancer" was widely circulated and largely used as the reverse side of the program of cancer meetings. Approximately 22,150 of these were distributed.

"A Few Case Histories" is a small folder prepared by a member of the Pondville Hospital staff, which was mailed with a letter to each physician in the State.

Bill enclosures, carrying a few danger signals of cancer, with a list of cancer clinics in given sections of the State, and similar "stuffers" for pay envelope enclosure were prepared from carefully selected material and were given wide circulation through the co-öperation of public utility, mercantile, and industrial concerns.

Posters, flyers for announcing the program, trolley car placards, and billboard posters were also used. In addition to this specially prepared material, an unusually large amount of standard literature published by the American Society for the Control of Cancer, the State Department of Public Health and others was used.

POSTERS

Large posters entitled "Danger Signals", listing the four leading signs of cancer, were loaned for use in health centers and similar places.

Smaller posters to the number of 100 in the same style were given to industrial plants for use in welfare departments and on health bulletin boards. These were frequently used in combination with a card of the same size announcing local clinics.

Printed cards, in size 12″ by 15″, were distributed among larger industrial plants giving information about clinics.

Charts showing the attendance at each of the cancer clinics and the reason why patients came to the clinic were loaned to the local committees for use in information booths.

MOTION PICTURES

Three copies of the film "A Fortunate Accident" and two copies of "The Reward of Courage" were in circulation throughout the campaign. We are indebted to the American Society for the Control of Cancer for the loan of four of these copies.

A "creeper" announcing the campaign and its purpose was shown in twenty-five motion picture houses in Boston and Springfield.

Slides in sets of four each, giving condensed information regarding early signs of cancer, were shown in fourteen motion picture houses.

GROUPS REACHED

Churches. A letter was addressed to each of the Protestant Clergymen in the Boston Federation of Churches and to the president of each one of the thirty federations outside Boston, asking that pulpit announcement of the campaign be made on Sunday, April 22nd; that requests for speakers be made for church groups; and that announcement of the campaign be made in church bulletins. Copies of the same pulpit notice were also sent to the newspapers on the regular list of 200. Catholic churches were reached through eminent leaders in the church. Nine talks were given before church organizations in response to the above request. Notice was given in a number of church bulletins. A number of patients reporting at the clinics stated that they learned of the clinic through notices given in churches.

Public and Private Agencies. A list of cancer clinics throughout the State was sent to each board of health, the president of each visiting nurse association, to each board of public welfare, and to family welfare agencies with the request that these organizations make it their responsibility to give information regarding cancer clinics whenever it is requested. These clinic lists were printed in several board of health monthly bulletins.

The Medical Profession. THE NEW ENGLAND JOURNAL OF MEDICINE gave generously of its space, both in preliminary announcement of the program and by giving over the pages of the JOURNAL for a special cancer number on April 26th. At this time articles appeared by Dr. Robert B. Greenough, Dr. David Cheever, Dr. George Gilbert Smith, Dr. Herbert L. Lombard, Dr. Carl R. Doering and Dr. Ernest M. Daland, as well as the addresses given by the Governor and others at the opening meeting in Symphony Hall.

Each District Medical Society was asked by Dr. Greenough as Chairman of the Committee on Cancer of the Massachusetts Medical Society, to hold a special meeting during the week of the campaign to consider some phase of the cancer problem. Of the eighteen district societies, fourteen held special cancer sessions, some individually, others in joint session with other district societies.

Special clinics were conducted at the Palmer Memorial Hospital, Pondville Hospital, Lowell General Hospital, Worcester Memorial Hospital, North Adams Hospital, the Wesson Memorial

Hospital at Springfield, the Lynn Hospital and the Franklin County Hospital at Greenfield (opening of the cancer clinic).

A letter addressed to the medical profession was sent by the committee with the folder "A Few Case Histories" to all physicians in the State at the time of the opening of the campaign.

Speakers. An outline "Facts and Theories Regarding Cancer" was prepared by the Department for the guidance of the speakers who had responded to the request to assist in the campaign.

Fifty-seven speakers addressed 129 groups, varying in number from 20 to 1900. The total audience reached, as estimated by the various speakers, was approximately 12,000. This estimate makes no account of the number reached by local committees in individual communities. Among the speakers were the Governor and the Mayor of Boston both of whom took part in the opening meeting in Symphony Hall. The mayors of other cities spoke at their local meetings and notable out-of-state physicians gave generously of their time and energy. Requests for speakers were met as follows:

Home Extension Service Groups	21
Men's Luncheon Clubs	17
Industrial Groups	14
Women's Clubs	10
School Centers (in Boston)	9
Church Groups	9
Others	49

Radio. Stations WBZ and WEEI made a generous offer of their resources. Daily twelve-minute talks, two afternoon talks, and announcements each evening were given through the former station; one evening talk was made through the latter, and daily announcements in connection with news flashes were made from both. One talk was given from Station WBET. Station WTAG in Worcester also gave time for a radio talk and for announcements by members of the Worcester Cancer Committee. WMES offered its service which was taken advantage of after the campaign week.

Industries. The co-öperation of 231 of the larger industrial plants and public utility companies was sought. The approach to a number of the firms having headquarters in Boston was made by a member of the Boston Cancer Education Committee who has had wide contact with business houses. Outside Boston visits were made to the establishments by a member of the staff of the Department.

In response to these requests there were circulated: 287,700 bill enclosures; 141,077 pay envelope slips; 725 small posters. Thirty articles were published in the same number of house organs. Fourteen speakers addressed industrial groups.

Public Libraries. In Boston the Public Library issued a special bulletin list of books and pamphlets on cancer, adapted for popular reading and approved by the Department. These have been placed on the shelves of the library for permanent use. In New Bedford there is available in the reference room of the Free Public Library a number of publications on the subject of cancer. The Fitchburg Public Library placed "Cancer and the Public" by Charles P. Childe on its shelves.

RESULTS

Results should of course be estimated in terms of persons influenced to seek service sufficiently early to effect cure. No short term revue such as this can give any such final estimate. For the present we must content ourselves with a study of the number of persons seeking advice as a result of the campaign.

The following questionnaire was sent to all physicians in the State:

1. Number of individuals WITH CANCER who consulted you during the month of May as a result of the Cancer Campaign.
2. Number of individuals WITHOUT CANCER BUT WITH SOME DEFINITE DISEASE who consulted you during the month of May as a result of the Cancer Campaign.
3. Number of individuals WITHOUT CANCER OR OTHER DISEASE who consulted you during the month of May as a result of the Cancer Campaign.
4. What is your opinion of the value of such campaigns.

(Note: Do not include cases which have been seen at any Cancer Clinic)

The non-state-aided cancer clinics were asked to report their total attendance, number with cancer or suspicious cancer and pre-cancerous conditions for the weeks preceding, during and following the campaign. The state-aided clinic records were tabulated for attendance and diagnosis for the two weeks preceding the campaign, for the campaign week with the week following, and for the two ensuing weeks. Intervals of two weeks were chosen after studying the clinic records from March 1, 1928, to June 1, 1928, as the two weeks prior to the campaign were typical of all the others and the weeks following the campaign were similar to those preceding.

About twenty-five per cent. of the Massachusetts physicians responded to the questionnaire (1479). These physicians examined 438 individuals with cancer out of a total of 3086 (14 per cent.), as a result of the campaign. If the physicians who answered the questionnaire were a fair sample of the Massachusetts medical profession, approximately 1780 persons had cancer of some 12,200 who consulted doctors as a result of the campaign.

This does not include those who attended cancer clinics. It may be said that as those seeing the most cancer are more likely to answer the questionnaire and that they do not give a fair picture of the experience of all the doctors of the state. However, as many of those answering reported no cases from the campaign, the

1400 doctors may not be as unrepresentative as might at first appear.

The following table shows the number of individuals with cancer who consulted physicians as a result of the campaign:

	No. of Doctors	Cancer Cases	Cases per Doctor	Per Cent.
Boston Doctors	466	155	.33	14.5
Doctors in Clinic Cities	339	126	.37	14.5
Doctors in Other Cities	498	157	.32	13.7
Total	1303	438	.34	14.2
Doctors not in Practice	103			
Doctors in Other Work	73			
Grand Total	1479			

An opinion as to the value of the campaign was expressed by 1132 physicians. Twenty-six (2.3 per cent.) considered the campaign of no value; thirty-eight (3.4 per cent.) considered it harmful; 108 (9.5 per cent.) were doubtful as to its value, and 960 (84.7 per cent.) considered it worth while.

The following table shows the clinic attendance before, during and after the Cancer Campaign. The number of cancer cases seen in the clinics increased during the campaign weeks and rapidly decreased thereafter. The per cent. with operable cancer was about the same during the campaign weeks as before, which fact indicates that phobia was not appreciably increased.

The records from the non-state-aided cancer clinics show conditions similar to those of the state-aided ones. There was a large increase in the attendance during the two weeks of the campaign which was followed by a decline nearly to normal during the following two weeks. However, that the decline was not to below normal may be credited to sustained interest, since the marked increase in attendance during and just after the campaign might be expected to drain somewhat the future "normal" supply for the clinics.

SUMMARY

About one-fourth of the physicians of Massachusetts reported that 3086 individuals consulted them during May as a result of the cancer campaign. These were in excess of the cases normally visiting the doctors irrespective of the campaign. Of these, 438 had cancer and 1427 had some other disease. If the group that reported is a fair sample of Massachusetts physicians, over 12,000 persons consulted doctors as a result of the campaign, 14 per cent. (1680) of whom had cancer.

The per cent. of cancers in the clinics prior to the campaign and during the campaign showed little difference. This indicates that phobia was not aggravated by the campaign.

Eighty-five per cent. of the doctors who reported stated that in their opinion such a campaign was valuable.

In the clinic cities the number of individuals going to private physicians was larger than in other cities. This was true both for total attendance and for cancer cases, about twelve cancer cases going to private physicians as a result of the campaign for every one who went to the

CASES ATTENDING STATE-AIDED CANCER CLINICS

	Pre-Campaign (two weeks)			Campaign (two weeks)			Post-Campaign (two weeks)		
	Cases	Per Cent.		Cases	Per Cent.		Cases	Per Cent.	
Operable cancer—chance for cure	15	15.2	27.3	31	15.2	26.4	23	18.1	24.5
Inoperable cancer	1	1.0		6	2.9		4	3.2	
Operable cancer—palliative only	2	2.0		7	3.4		1	0.8	
Precancerous condition	9	9.1		7	3.4		3	2.4	
Post-operative cancer—no evidence of recurrence	0	0		3	1.5		0	0	
Benign tumor	10	10.1		16	7.8		6	4.7	
Other conditions	48	48.5		90	44.1		61	48.0	
Normal	4	4.0		12	5.9		8	6.3	
Deferred	10	10.1		32	15.7		21	16.5	
Total	99	100.0		204	99.9		127	100.0	

CASES ATTENDING NON-STATE-AIDED CANCER CLINICS

	Pre-Campaign (two weeks)		Campaign (two weeks)		Post-Campaign (two weeks)	
	Cases	Per Cent.	Cases	Per Cent.	Cases	Per Cent.
Cancer or suspicious cancer	78	33.9	116	31.5	67	26.4
Pre-cancerous condition	19	8.3	49	13.3	36	14.2
Non-cancer	133	57.8	203	55.1	151	59.4
Total	230	100.0	368	99.9	254	100.0

clinic. When the total clinic attendance is compared with the attendance in the physicians' offices as a result of the campaign, twenty-two individuals went to doctors to every one that went to the clinic. We have no explanation of why the proportion of cases with cancer was two or more times as great among those attending clinics as among those going to private offices unless it is that the non-cancerous are to a certain extent inhibited from attending "cancer" clinics.

During the week of the campaign and the one following, the clinic attendance was more than double, but it rapidly subsided to normal. This is striking evidence of the short duration of the intensive interest in cancer stimulated by the campaign. Whether or not such short term interest is desirable or not is a matter for wide difference of opinion.

However, with the State-wide local medical and education committees, education will be continued throughout the year so that the criticism of apparent indifference once the "week" is over will be avoided, and continued benefit should be felt from information disseminated to the profession and the public.

The total extra cost to the three organizations was approximately $4,500.

GOLD MEDAL AWARDED DOCTOR EDWARD FRANCIS FOR WORK ON TULARAEMIA, THE NEW AMERICAN DISEASE

A gold medal was presented to Dr. Edward Francis, of the United States Public Health Service, Washington, D. C., by the American Medical Association during the meeting recently held in Minneapolis, Minn. The committee on awards considered the research work on tularaemia, a new disease of man, as the most important medical work of the year. Recognizing Dr. Francis as the nation's outstanding authority on this disease which has perplexed science for several years, the committee, in judging his work on the basis of originality, made the statement that the medal was being awarded to him for his thorough and important scientific contributions to the knowledge of tularaemia.

Tularaemia is primarily an epizootic of wild rabbits and is caused by *Bacterium tularense*, which affects the liver and spleen, producing decay of the tissue cells in these organs shown by innumerable white spots from the size of a pin-point to that of a pinhead to be studded over this surface and resulting in death. Of the wild rabbits offered for sale in the Washington, D. C., market Dr. Francis examined the livers of 1,000 and found 10, or one per cent., to be infected with virulent *Bacterium tularense*.

Man readily inoculates himself with the disease while dressing rabbits, the infection passing from the rabbit's liver through some wound on his hand, resulting in an ulcer on the hand, enlarged glands at the elbow or in the arm pit, and fever which confines him to bed for two or three weeks.

Cooks, hunters, housewives, and market men are often infected in November, December or January, when, owing to relaxation of the game laws, it is permitted to hunt wild cotton tail rabbits for food.

Persons who skin and cut up jack rabbits for bait, or for the market, frequently become infected.

Tularaemia is "made in America" and has been staged from start to finish by an all-American cast. The discovery of a new disease is an important milestone in medical history. In the history of human medicine there is only one instance in which American investigators alone have discovered a disease of man, isolating its causative agent, determining its sources of infection and its modes of transmission to man, describing its symptomatology and pathology, and otherwise elucidating the many essential problems connected with the complete knowledge of a disease—that instance is the story of tularaemia.

The disease was first discovered in a ground squirrel in Tulare County, California, in 1910, by Dr. G. W. McCoy, of the United States Public Health Service. It became engrafted into the jack-rabbit population of the West, and then, as a disease of wild rabbits and of man it advanced steadily across the Continent, invading State after State until now, in 1928, there remains only a solid block of six uninvaded States composed of the New England group.

Although a new disease of man, tularaemia has now been recognized in 42 States of the United States, in the District of Columbia, and in Japan, but in no other country. Of 614 reported cases, 23 have terminated in death.

Dr. Francis himself fell a victim to tularaemia while studying his first case of the disease in Utah. He is now devoting himself to its prevention and cure.

Prevention is the key-note of modern medicine. Keep your bare hands out of a wild rabbit—one per cent of them are infected with tularaemia. Rabbit meat, thoroughly cooked, is harmless for food, because a temperature of 133° F. kills the infection. Rubber gloves afford complete protection to those who must dress wild rabbits.

Beware of the wild rabbit which the cat or dog has caught—or which a boy has killed with a club—it is probably a sick rabbit. A warning to the poor sportsman is necessary. He should not shoot the rabbit that is on the point of his gun. Let him take his rabbits on the run at twenty-five feet distant and the chances will be lessened that the rabbits he bags will be sick with tularaemia.

The women of the country are coming to the rescue. They are telling their sportsman husbands to bring home the birds, but to let the rabbits lie as they fall—"Don't bring them home." The disease is new, but the warning is 5,000 years old. Read Leviticus, chapter eleven, verses four to eight: "The flesh of the hare shall ye not eat, and its carcass shall ye not touch; they are unclean to you.—*United States Public Health Service.*

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

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CASE 14281
JAUNDICE AND ASCITES IN A BOY OF EIGHTEEN

MEDICAL DEPARTMENT

An American schoolboy of eighteen entered the hospital September 28 with a diagnosis by his physician of cirrhosis of the liver.

A year and a half before admission when in apparently good health he woke up at night with a very sharp pain in the abdomen localized below the umbilicus, more on the right. It doubled him up for twelve minutes, then gradually wore off. He stayed in bed two days. There were no clay colored stools or jaundice. From thirteen to seven months before admission he had gas (heartburn) relieved by white powders given by his physician. A year before admission after a cold in the head a physician found a slight infection of the gall-bladder with some jaundice. He had dull generalized headache when walking, but no other pain. He was in bed a week and recovered completely in three weeks. His urine was dark. During the next six months he was not quite in his usual health and had three similar attacks, two of them just before the holidays, when he had been eating much candy. As the last attack was subsiding, six months before admission, he went to a physician, who found a severe bronchitis which lasted three weeks. There was also an acute ear. The liver was somewhat enlarged. There was more or less continuous right sided pain along the eighth and ninth ribs relieved by lying on his right side, possibly pleurisy. He vomited once. The vomitus contained dark material resembling old blood. At the onset of the next attack, three months before admission, his physician found an enlarged liver, ascites and edema of the feet and legs. The temperature was 101° to 105° for three weeks. During this period there were night sweats. All the symptoms then disappeared. Two weeks before admission the patient had a severe cold followed by symptoms similar to those of the last attack. These symptoms persisted. With the cold he had large glands in the neck which had subsided at admission. He had been on a skim milk diet with sodium phosphate enough to keep his movements watery. He had vomited only once; blood in the vomitus was questionable.

One grandfather died of cancer. The family history is otherwise not important.

The patient had had no serious diseases, but had always been in perfect health and very strong until the present illness. He had a questionable light attack of influenza nine years before admission. Six years before admission he swallowed a nail which gave no symptoms and was never recovered. Six months before admission he had the questionable pleurisy mentioned above. Four months before admission he had a purulent discharge from the right ear which cleared up. He urinated once or twice at night. At admission his mouth was sore, possibly from a wisdom tooth. Six months ago he weighed 140 pounds; his present weight was 142.

Clinical examination showed a weak, wasted, ill-nourished boy with dry and jaundiced skin, jaundiced sclerae and sparse hair, unable to sit up. The tonsils were enlarged, cryptic, and filled with cellular débris. There were pea-sized axillary glands. The chest was flat. The ribs were prominent. The apex impulse of the heart was not seen or felt. There was no enlargement to percussion. The sounds and action were normal. The pulmonic second sound was accentuated. There was a systolic murmur in the left third interspace. Blood pressure 100/50 and 30. There was some dullness at the bases of the lungs behind. The abdomen was tense and shining, with shifting dullness, very marked fluid wave and an air bubble. The liver edge was felt by ballottement three finger-breadths down at least, slightly tender. The spleen was felt in the same way three fingers down. There was slight umbilical hernia. The scrotum was filled with fluid and enlarged to the size of an orange. Both legs showed pitting edema to the thighs. The pupils were normal. The knee-jerks were obtained on reinforcement.

Before operation urine normal in amount, specific gravity 1.040 to 1.020, cloudy at 4 of 6 examinations, alkaline at all, the slightest possible trace to a very slight trace of albumin at 5, bile in 6 of 7 specimens, 0 to 1 leukocytes and 0 to 1 red cell per field in one of six specimens of sediment. Blood: 800 to 3,450 leukocytes, 52 per cent. polymorphonuclears, 24 per cent. lymphocytes, 12 per cent. large mononuclears, 12 per cent. unclassified. (Differential on a basis of 25 cells.) Hemoglobin 45 to 55 per cent., 3,000,000 to 2,600,000 to 3,100,000 reds, achromia, slight variation in size and shape, no stippling or polychromatophilia. Platelets decreased. Reticulated cells 1 per cent. Icteric index 50 at admission, 20 October 6 and 12, 18 October 17. Bleeding time 8½ minutes to 4½ minutes. Clotting time 7 to 9 minutes. Retraction normal. Fragility test began at 44, complete at 28. Wassermann negative. Van den Bergh test direct, positive, quantitative 8.8 milligrams per 100 cubic centimeters. Rosenthal bromsulphalein test: after 30 minutes 20 per cent. Cholesterol (serum), .084 per cent. Stools: guaiac negative at 3 examinations, bile indefinite at 2.

Until October 12 temperature 97° to 101°, then 98° to 103° until operation. Before operation pulse 78 to 121, respirations 12 to 32.

The day of admission an abdominal tap gave 4,000 cubic centimeters of clear bile stained fluid, specific gravity 1.008, 500 red cells and 100 leukocytes, mostly lymphocytes; culture, no growth. Immediately after the tap the liver edge was felt two-thirds of the way down to the umbilicus, extending well to the left. The edge was sharp and slightly tender, and appeared grossly irregular, as if the liver were lobulated. The spleen was felt 2 centimeters below the costal margin. The patient was more comfortable after the tap.

An X-ray plate taken October 4 after a barium meal showed a very large gastric outline. The stomach was apparently distended by gas. The outline of the transverse colon was also visible and appeared to be filled with gas. The diaphragm was high on both sides. Just above it on the right there was a triangular area of increased density with very sharply defined margins, possibly an area of atelectasis of the lung. Otherwise the examination of the chest was negative. There was nothing unusual in the appearance of the heart shadow.

The jaundice cleared rapidly and by October 7 was nearly gone. Distention was now the chief complaint. The fluid in the abdomen reaccumulated. The patient was symptomatically better.

October 21 a fluoroscopic examination of the stomach without barium showed a large amount of gas in this region. The colon was also distended with gas as in the previous examination.

After a number of consultations operation was done October 22. After the second day the temperature came down to normal. There was moderate distention relieved by enemas. The wound was in good condition. The patient however did not look very well. The temperature was 100° October 29 and after this ranged between 99.2° and 101.8°. October 31 the patient was much worse, irrational part of the time, with very poor nutrition. Intravenous glucose was given without result.

November 1 blood examination showed 9900 leukocytes, hemoglobin 60 per cent., 3,300,000 red cells fairly well filled with hemoglobin, considerable variation in size, but only slight poikilocytosis; platelets normal. A count of about 500 cells showed no lymphocytes, 99 per cent. polymorphonuclears, a few immature and a few old, but no more than a normal smear would show. No smudges were made out.

November 2 the patient died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

Cirrhosis of the liver is a very surprising diagnosis at that age. What do you think of at this age or younger that looks like cirrhosis of the liver, but is not?

Student: Banti's disease. Syphilis.

Dr. Cabot: Either of those. If you have advanced Banti's disease you have cirrhosis of the liver. Anything else?

A Student: Acute yellow atrophy?

Dr. Cabot: It does occur. It is not common. Gall-bladder trouble would not give ascites. That is probably what they had in mind.

A Student: Syphilis?

Dr. Cabot: That is possible.

A Student: Tuberculosis?

Dr. Cabot: Tuberculous peritonitis is possible, yes. There is also a case I happen to remember here, a chronic pericarditis working through the diaphragm and to the capsule of the liver, with actual cirrhosis.

"He swallowed a nail which gave no symptoms and was never recovered." That is what you would expect. There is no reason why it should not pass through without being noticed in any way.

Some of that increased weight was presumably due to ascites and edema.

The main thing we have to bear in mind as we come to the physical examination is that the boy has had ascites, apparently enlarged liver and some previous attacks with fever and symptoms that sound like gall-bladder inflammation. Of course gall-bladder inflammation cannot account for enlarged liver and ascites. He may perfectly well have had both, but he could not have the latter as a result of the former. No one has said anything about the spleen, or of the possibility of the only type of neoplasm seen at this age often, that is the leukemic. He had some enlargement of glands in the neck, but it subsided. We must remember the possibility of tuberculous peritonitis. But I am quite at sea prior to the physical examination.

NOTES ON THE PHYSICAL EXAMINATION

That systolic murmur does not mean anything in a boy with this clinical picture.

With a big liver dullness at the bases of the lungs is probably due to a high diaphragm, not to anything wrong in the chest.

"The abdomen was tense and shining, with shifting dullness, very marked fluid wave and an air bubble," that is, the intestines floating up in the epigastrium.

I do not see anything about sugar in the urine report, but I assume that it is absent. Of course in an emaciated boy we naturally would wonder about sugar. But it would have been put down if it had shown in the urine.

The blood shows a secondary anemia. The chief thing about the blood is the extraordinarily low leukocyte count.

The lungs certainly look all right in this X-ray. The diaphragm is always higher on the right than on the left; it seems abnormally high

on both sides, which is natural enough in his condition.

DIFFERENTIAL DIAGNOSIS

It seems to me we have to say he has a cirrhosis of the liver, as they said in the beginning. I do not know what else would give such a chronic portal obstruction as we have evidence of here. The tap fluid is enough to throw out tuberculous peritonitis. After such a tap if there were any malignant disease we ought to be able to feel it.

We are not given any evidence of a chronic pericarditis. When you put these things aside I do not know anything that is left to cause such an ascites as this except a chronic interstitial process in the liver. What type can it be? I do not see any probability at all that it is the usual type which most of us associate with alcohol. Syphilis of course comes to our minds when we hear that the liver edge was lobulated. But we have nothing else to back up that diagnosis. Syphilis of the liver at this age is unusual. We do not usually have so much ascites. We often do not have jaundice, and I never knew it to kill anybody without other troubles.

Acute yellow atrophy or subacute yellow atrophy, toxic hepatitis, I can not exclude. We seem to have a big liver, and that, if it is correct, would exclude it. There may be stages of a toxic hepatitis without any diminution in the size of the liver. But we should not expect it to go on to death without diminution in its size. That is the chief evidence against it; but I do not feel able to exclude it. He has had two infections, one in the abdomen and one in the ear. It might be an infectious type of cirrhosis starting from a cholangitis and spreading up from the bile passages.

His anemia is an outstanding thing. We have to consider this as a hemolytic type of anemia and the hemolytic type of jaundice, connected with some abnormal activities in the spleen. I have not much doubt that the operation done was splenectomy for the removal of trouble supposed to be of that type. Between that and the infectious type of cirrhosis I do not know any way to make a clear differentiation.

Banti's disease is one of the most cloudy clinical entities that we have. I think the diagnosis cannot be made unless we prove first that we have an enlarged spleen and later a cirrhotic liver. It cannot be excluded here, but I cannot say anything more in its favor.

A STUDENT: How about the pitting edema of the legs and ankles? It does not fit in with cirrhosis of the liver, does it?

DR. CABOT: Yes, perfectly well. Almost all the cases of long-standing cirrhosis have sooner or later edema of the legs and feet. The circulation of the legs has to be interfered with indirectly. It is local interference, not depending upon heart weakness.

A STUDENT: How about Hodgkin's disease?

DR. CABOT: I see no reason to consider Hodgkin's disease. We hear of cases that are confined to the spleen and liver, but we should have no evidence outside that, especially when we have such a very low white count. I should think we had better not. I have no idea how to explain that differential count of 99 per cent. of polymorphonuclears. I have never seen so high a count. I feel rather skeptical.

A STUDENT: Couldn't that transparent area in the X-ray be due to enlarged glands in the hilus obstructing return of the blood through the vena cava?

DR. CABOT: In the absence of an X-ray expert I can only say that it does not look like glands to me. I may be wrong.

A STUDENT: Could it be the heart?

DR. CABOT: I think not. I believe I can see the heart in its ordinary position. It certainly cannot be a dilated heart I think.

A STUDENT: Do you think it could be atelectasis?

DR. CABOT: That is what the X-ray man said, and I am not expert enough to say better.

Dr. Mallory shows me the temperature chart with a big swing for a while just before the patient was operated on.

A STUDENT: That is common in Hodgkin's, isn't it?

DR. CABOT: Yes.

A STUDENT: How about the normal fragility?

DR. CABOT: We do not expect it.

A STUDENT: What would you call the immediate cause of death?

DR. CABOT: I am not certain. Of course with an operation of this kind done on such a debilitated patient we should naturally think of sepsis.

A STUDENT: Would the negative Wassermann help in the diagnosis of syphilis here?

DR. CABOT: Yes; it does not exclude it, but it helps.

A STUDENT: Isn't this large amount of ascites against biliary cirrhosis?

DR. CABOT: I tried not to mention that particular type of cirrhosis. I do not know anything about it, that is, the so-called Hanot's cirrhosis. But you get ascites, as far as I know, in some of the infectious types of cirrhosis, which are what I am thinking of here.

A STUDENT: How do you rule out malignant disease of the liver?

DR. CABOT: I have never seen it at eighteen.

A STUDENT: How about melanotic sarcoma?

DR. CABOT: You have to have some evidence of melanotic tumor somewhere. I never heard of anything of that kind in one so young either. I feel very uncertain about the case, but one of the few things that I do feel sure of is that he has no malignant disease in the liver.

DR. TRACY B. MALLORY: Infectious cirrhosis was their diagnosis.

PRE-OPERATIVE DIAGNOSIS

Infectious jaundice.

OPERATION

The abdomen was found to be filled with clear fluid. The liver was very much enlarged, perfectly smooth and instead of being dark was rather speckled in appearance and lighter in color than normal. A small piece was taken out for microscopic study. The spleen was much enlarged, about eight inches in length, much thickened and also felt smooth. The gall-bladder was much thickened and edematous. In the retroperitoneal tissue above the pancreas there seemed to be more or less thickening or edema, as the tissue was thick enough to prevent the feeling of the normal structures. A tube was put into the gall-bladder.

FURTHER DISCUSSION

DR. CABOT: I do not see that until the microscopic examination is made we know any more than we did before.

DR. MALLORY: The microscopic findings were a complete surprise to everybody. The liver showed a very marked infiltration around all the portal areas of large, immature, abnormal lymphocytes showing very numerous mitoses. The histologic appearances were typical of lymphatic leukemia. I think the only possible diagnosis is an aleukemic lymphatic leukemia.

DR. CABOT: Were there similar changes in the other organs?

DR. MALLORY: We unfortunately did not have a post-mortem. The histologic appearance was very characteristic. I think there is no possibility of doubt.

DR. CABOT: Did you ever see a case like this before—with ascites and nothing else?

DR. MALLORY: No, I never did.

A STUDENT: Was the spleen removed at operation?

DR. CABOT: No, I do not see that they did anything.

DR. MALLORY: At the time of operation they still believed that it was an infectious cirrhosis and therefore drained the gall-bladder.

A STUDENT: Why were there no lymphocytes at all seen in the blood smear?

DR. MALLORY: In leukemia of this type the lymphocytes are very abnormal. I suppose the explanation is that they are not sufficiently differentiated to live in the circulating blood stream. They are essentially tumor cells; no normal lymphocytes are being produced at all.

DR. CABOT: Should you assume that we should have found something in the marrow?

DR. MALLORY: Yes. I believe all the organs would be similarly infiltrated.

HISTOLOGIC DIAGNOSIS

Lymphoblastoma.

CASE 14282

FEVER, WEAKNESS AND PAIN IN THE RIGHT AXILLA

MEDICAL AND SURGICAL DEPARTMENTS

First admission. A married American woman fifty-nine years old, a cleaner, came for erysipelas of the face, which cleared up in ten days.

Second admission. November 19, seven months later, she entered for study. The chief complaints were weakness and a feeling of fatigue. Because of her drowsiness and slight deafness the history was not satisfactory.

For a week she had felt tired. Three days before admission she had headache, vomiting, chill, night sweat and constipation.

Her mother died of consumption.

The patient had had several attacks of "grippe" in the past,—fever, cough, sputum, bed for six weeks.

Clinical examination showed a drowsy, feverish woman. Dullness and increased tactile fremitus and whispered voice at both tops, with bronchovesicular breathing. Abdomen slightly distended. Palpable liver and spleen; spleen hard for typhoid.

The urine showed evidence of a pyelitis which cleared up in the hospital. No tubercle bacilli were found on special search on three occasions. Blood: 10,000 to 23,000 leukocytes, polymorphonuclears 82 to 50 per cent. At entrance hemoglobin 80 per cent., smear normal except for apparent decrease in the number of platelets. Later slight achromia, variation in size and shape, and increase of platelets. Reticulated cells 0.5 per cent. Wassermann negative. Two blood cultures negative; one showed bacillus epidermidis communis in one of two flasks. Widal negative. Non-protein nitrogen normal. Sputum negative.

Temperature for the first week 100.7° to 105.1°, rectal, with a sharp drop to normal November 25 and 26, later 98° to 101.4°. Pulse 79 to 122. Respirations 17 to 37.

X-ray examination November 25 showed the left chest less radiant than the right. The outline of the diaphragm was very indistinct on this side. The changes were more marked toward the base. In both upper lobes there were many small sharply defined dense shadows scattered along the course of the lung markings and extending well into the lung apices. The heart shadow was somewhat enlarged. The plate was not entirely satisfactory on account of motion.

The patient had persistent nausea and vomiting. On a liquid diet the spleen became smaller and softer than at entrance. November 24 there was massive consolidation in the left midchest posteriorly, much increased and nasal voice sound, tubular breathing. Distention was marked and persistent, though the abdomen was soft and not tender. November 26 she was stuporous. After the drop in temperature, pulse

and respirations, she felt better and the distention was less, though she was still somewhat stuporous. There was a slight friction rub in the left lower chest. November 28 there were many râles. Two days later she complained of pain in the left lower chest, where a loud friction rub persisted. The temperature, pulse and respirations were again elevated. December 2 there was no friction rub. December 5 there was pain in the chest and slight friction rub just to the right of the sternum in the fifth interspace.

The patient showed such unusual lethargy throughout that a neurologist asked for a lumbar puncture. On its evidence he could make no diagnosis. He found the fundi normal, the right knee-jerk absent. About December 15 a lung specialist found no abnormality except slight dullness with increased whispered voice, especially at the left apex. An ear specialist found no cause for fever in the ears. A series of lesions on the tongue showed no mycelium.

The patient continued to have a little unproductive cough and constant temperature without change in the chest signs. Another X-ray examination showed practically the same appearances as before. There was no evidence of a pathological process in the left base. In the region of the aortic arch there was a ring-like shadow which it was thought probably represented calcification in the aorta. The patient made steady improvement. December 31 she was discharged.

History of interval. After leaving the hospital she was perfectly well for a year except for an occasional bad cold. February 8, thirteen months after her discharge, she developed severe headache, was feverish, had a sore throat, loss of voice and slight productive cough.

Third admission, February 11.

Clinical examination showed a poorly nourished woman with slightly rapid rasping breathing and hoarse voice. Throat red. Tonsils enlarged and red. Dullness at both apices extending 3 to 4 centimeters below the clavicle. Increased voice and breath sounds. No râles. Apex impulse of the heart felt 9 centimeters to the left of midsternum in the fifth space, 1.5 centimeters outside the midclavicular line, coinciding with the left border of dullness; supracardiac dullness 7 centimeters, right border 5. Sounds and action normal. Aortic second sound and pulmonic second equal. A very soft systolic murmur at the apex. Artery walls thickened and tortuous. Blood pressure 150/80. Splenic dullness began in the eighth interspace. Spleen descended 1 to 2 centimeters below the costal margin, not hard. Right kidney could be held between the fingers.

Urine normal in amount, specific gravity 1.018 to 1.007. Sediment of two catheter specimens showed leukocytes and occasional red cells. Culture of one showed profuse growth of colon-like bacilli. Renal function 25 to 35 per cent. Blood: 29,000 to 10,500 leukocytes, 72 per cent. polymorphonuclears, hemoglobin 75 per cent., reds 4,600,000, slight central achromia, smear otherwise normal. Wassermann negative. Nonprotein nitrogen 36 milligrams. Two throat cultures showed streptococci, no Klebs-Loeffler.

Temperature 102.5° to 98.9° until February 22, afterwards 97.9° to 100.4°. Pulse 110 to 73. Respirations 20 to 28.

X-ray examination showed extreme calcification of the arteries of the hands and wrists. Both antra were small and slightly increased in density. Their outline was rather irregular. The frontal sinuses appeared normal. Both lung fields showed numerous small dense calcified areas, especially in the upper portions. There was considerable hilus fibrosis, more marked at the right base; no definite evidence of any active disease.

The patient was lethargic, as she was in her previous illness. By February 23 the temperature was flat, the chest clear. Slight cough persisted. March 1 she was discharged.

Fourth admission. February 16, three years after her last discharge, she entered complaining of sharp pain beneath and to the right of her right breast coming on suddenly twenty-four hours before admission. With it she felt weak and tired. No history of the interval was obtained.

Clinical examination showed a poorly nourished woman looking ill, breathing somewhat rapidly. Skin warm and dry. Lips, tongue and pharynx very dry. Heart showed no enlargement. Rhythm regular. Rate rapid. Sounds of good quality. A systolic murmur over the precordia. Radials and brachials hardened and beaded. Blood pressure 115/80 to 163/70. Well defined friction rub in the right axilla. Breath sounds increased in intensity at both apices behind,—not bronchial. No râles. Expansion limited. Abdomen slightly resistant to pressure throughout. Spleen descended from 2 to 4 centimeters on inspiration. Fundi: hyperopia; discs and vessels normal. Pupils and reflexes normal.

X-ray February 19 showed both lung fields less radiant than normal. The diaphragm was low, normal in outline. The costophrenic angles were clear. There was an area of dullness at the right base and cardiophrenic angle fading indefinitely toward the periphery. There was generalized calcification in the aorta with several calcified plaques.

Temperature 97° to 103.5°, rectal. Pulse 85 to 155. Respirations 17 to 60.

Amount of urine 23 to 82 ounces, specific gravity 1.004 to 1.024, a trace to a very slight trace of albumin at 5 of 14 examinations, a faint trace of sugar once. Cultures of two catheter specimens showed colon bacillus. Precipitation test on urine showed Type I pneumococcus. Blood: 20,100 to 9,800 leukocytes, polymorphonuclears 86 per cent., hemoglobin 75

per cent., reds 5,392,000 to 4,672,000, smear normal. Stool and vomitus negative. Wassermann negative. Non-protein nitrogen 29 milligrams. Icteric index 6.

February 19 there was slight tenderness and a friction rub in the lower right chest in front and dullness with diminished breath sounds, egophony and questionable tactile fremitus in the right lower back. The first heart sound was loud, high and short. There was an occasional extrasystole, no murmur. The following morning the visiting physician found signs of consolidation at the right base. There was some distention, increased the next day. Her bowels moved on both days.

February 25 she looked and felt better. The abdomen was less distended. She continued to make symptomatic improvement until March 4.

An elevation of temperature to 102° on March 1 and 3 was not accounted for. March 6 the area of dullness previously noted at the right base had increased considerably in size and now rose as high as the seventh rib posteriorly. The diaphragm was faintly visualized through this dullness. March 8 the right lower back showed dullness, diminished tactile fremitus and breath sounds and nasal voice. There were a few râles in the right axilla.

March 10 a chest tap was done at the right base. 130 cubic centimeters of pea soup colored pus was withdrawn. Most of the cells were degenerated. Those identifiable were mostly polymorphonuclears with a few lymphocytes and large mononuclears.

March 11 a trocar thoracotomy was done in the seventh space and about two ounces of thin creamy pus removed. Culture showed pneumococcus Type II. March 14 there was still drainage of about one ounce every four hours. The temperature was still elevated. March 17 X-ray showed very little change in the chest since the last observation. The upper portion of the right lung field had cleared up slightly. March 28 another examination showed that the dullness at the base had possibly diminished a little. The patient did not void urine. April 3 there was a little redness around the wound. The temperature was 102° to 103°. By April 9 it was coming down. There was slight sepsis in the wound. Irrigations with Dakin's came clear after four or five washes. The patient seemed very weak and slept almost all the time. Examination of the chest was not remarkable except at the right base, where flatness and absent tactile fremitus extended well up to the midscapula. The pulse was high. The liver and spleen were enlarged, firm and not tender. April 12 X-ray examination showed that the process in the chest had cleared up considerably. There was now however dullness of the lower two-thirds of the left chest which obscured the outline of the diaphragm and was thought perhaps to be due to fluid, though consolidation could not be absolutely ruled out. April 14 the patient died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

She had the evidences of some acute infection.

"Spleen too hard for typhoid" is a very proper observation, though not an easy one to make; we find soft spleen in typhoid, hard spleen in malaria and most other diseases.

The signs in the lungs were those either of pneumonia or of tuberculosis. They were looking hard for tuberculosis.

We ought to know which percentage of polymorphonuclears goes with which count. We cannot tell in this way. The reticulated cells are about normal. The bacillus epidermis communis under these circumstances I suppose is nothing. In the blood, then, we have evidence of a slight anemia and nothing more except a possible leukocytosis.

From this report, without being experts in X-ray, tuberculosis is what we should say. There are some diseases in which a plate will do fairly well even though the patient moves, but for the question of tuberculosis we must have a patient who does not move at all; otherwise the lesions we need to have particularly clear are blurred.

In many patients with tuberculosis, nausea and vomiting are the striking things, even though all the trouble is in the lungs.

"The spleen became smaller and softer," but whether it had anything to do with a liquid diet I doubt.

On November 24 there was evidence of a consolidated lung.

In asking for a lumbar puncture they were thinking of the possibility of tuberculous meningitis.

Suppose we take this report of the specialist as a fact. If this is so, then what? Then it is not tuberculosis. Dullness never goes off like that, in a few weeks, in tuberculosis. If this is a fact then this must have been a wandering pneumonia going from lobe to lobe. I do not know what is coming, but on the findings thus far, with nothing now to be found by a competent observer except a little dullness, we cannot say tuberculosis; but X-ray evidence, the best we have about the chest, would tend to show that the man who made the stethoscope examination was wrong.

I should like to know what diagnosis they made. They cannot have made a diagnosis of tuberculosis. I guess they made a diagnosis of subacute or chronic pneumonia.

Miss Painter: The diagnosis was lobar pneumonia with an old tuberculous process at the apices.

DR. CABOT: They thought it was an old tuberculous process with an acute pneumonia which made all the more massive signs.

The condition at the third admission sounds like a bad cold.

They found signs which if they did not read the previous record would be quite important evidence of a healed tuberculous process. If they had read the previous record it would not be nearly so important. We are likely to find what we ought to find.

All the signs are consistent with an enlarged heart.

The splenic dullness was one interspace too high. We have never accounted for that spleen. We do not get a spleen in ordinary tuberculosis that is not miliary. We do not get it in any kind of pneumonia.

This note does not mean that the kidney is abnormal in any way.

They found evidence of an infectious process in the urinary tract, in the absence of any bladder symptoms presumably a pyelitis, and of a sore throat and slight anemia.

The calcification of her arteries is more than we expect in most people of sixty.

They were looking for a cause of fever in the antra.

The strikingly white spots scattered over the upper portion of both lungs in the X-ray plate seem to be similarly placed in all the plates,—healed tuberculosis.

They never found before why she was lethargic. Perhaps she was so by nature.

I do not believe that to-day she would have got into the hospital. She had a bad cold and they did find some leukocytes in the urine, but she was very slightly sick this time.

NOTES ON THE PHYSICAL EXAMINATION

Either they were wrong before or they are wrong now. The heart once enlarged is always enlarged. I guess it was enlarged, but it does not look so in these plates, I must acknowledge.

"Discs and vessels normal" is very surprising and shows how limited in area arteriosclerosis can be. She had this extreme arteriosclerosis mentioned in the hands and wrists and none at all in the retina.

There is a normal urine.

The non-protein nitrogen and the icteric index are normal.

The lung signs on the whole are those of fluid. They probably could have got more pus, but stopped. So we have, I suppose, a postpneumonic empyema on the right side.

We have never yet seen any reason to say why the liver and spleen were enlarged.

DIFFERENTIAL DIAGNOSIS

The first three entries give us nothing of importance in relation to our final study, except insofar as they reveal the healed tuberculosis at the apices of both lungs.

She comes in with pneumonia, she gets right sided postpneumonic empyema, we drain it, and she died, I suppose, of sepsis.

What is there going to be found in the left lung? Quite possibly there is going to be empyema there too. She was too sick for them to tap and find out. There may be serous effusion, there may be purulent effusion.

I am in doubt about the heart. We have had some observations tending to make us think it was enlarged. She may have acute endocarditis. It is the sort of case in which terminal endocarditis develops as part of a general septic process.

Why is the spleen enlarged? I do not know. Perhaps it is not.

A STUDENT: Could it be amyloid?

DR. CABOT: I have never seen amyloid in healed tuberculosis.

A STUDENT: She has a chronic pyelitis too.

DR. CABOT: I do not think that is enough. I do not believe it would cause an amyloid. It is a good suggestion, but I am voting against amyloid, because although the sort of thing she had in this last illness would produce amyloid in time, the things she had previously, so far as I know, would not.

I should say Dr. Mallory ought to find the remains of a pneumonia in the lungs, this proved empyema on the right and very possibly empyema on the left. It might also be solidification and might be serous effusion. The heart ought to be slightly enlarged, according to the balance of evidence. There may be acute endocarditis, but we have nothing to prove it. The rest of the body ought to be negative aside from this enlarged spleen, the cause of which I do not know.

INTERPRETATIONS OF X-RAYS

Second admission, November 27. The shadows in the left lung probably represent an old tuberculous infection. The appearance at the left base may be a beginning pneumonia.

Third admission, February 19. The calcified

areas in both lung fields represent healed tuberculosis.

Fourth admission, February 20. The findings are those of generalized fibrosis, arteriosclerosis and possibly a pneumonic process at the left base.

March 7. The findings suggest pneumonic consolidation.

REPORT OF LUNG CONSULTANT, SECOND ADMISSION

The history suggests a migratory pneumonia with an old tuberculosis of the left apex.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Lobar pneumonia.
Empyema.

DR. RICHARD C. CABOT'S DIAGNOSIS

Pneumonia.
Postpnuemonic empyema, right, possibly also left.
Possibly solidification or serous effusion, left.
Septicemia.
Slight hypertrophy of the heart.
Possibly acute endocarditis.

ANATOMIC DIAGNOSES

1. *Primary disease.*

   (Lobar pnuemonia.)

2. *Secondary or terminal lesions.*

   Pneumococcus pericarditis.
   Empyema.
   Bronchopneumonia.
   Arteriosclerosis.

3. *Historical landmarks.*

   Healed apical tuberculosis.
   Cholelithiasis.

DR. TRACY B. MALLORY: There are several points of considerable interest about this case. One is that it shows two of the common complications of lobar pneumonia. One is empyema, which was discovered and drained. The other is an acute purulent pericarditis which was not discovered and which presumably was the immediate cause of death. The lungs showed a few bronchopneumonic areas, presumably a recent process just in the last few days, the original pneumonia having cleared up. There also was extensive but apparently entirely healed tuberculosis at both apices.

I think the case is interesting also from the point of view of arteriosclerosis, since that was noted as extreme in the clinical record and was confirmed at post-mortem examination to be very extensive in the larger vessels, particularly the aorta, iliacs, and the coronary arteries. On the other hand, in the clinical record it was stated that the retinal arteries were negative, and microscopic examination of the kidneys shows that the arterioles there are entirely negative. That agrees with the observations of Walker and O'Hare in a large series of cases at the Peter Bent Brigham Hospital, that renal arteriosclerosis in the small vessels and arteriosclerosis of the retinal vessels commonly go together and commonly go with hypertension, which I think it is fair to say this case did not have. The diastolic pressure was never up and the heart was not enlarged. It weighed 280 grams.

The spleen was large. I have no explanation for it. It weighed 280 grams, and showed nothing remarkable histologically.

DR. CABOT: We cannot blame ourselves for not saying why it was big if Dr. Mallory cannot.

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THE LABORATORY DIAGNOSIS OF TUBERCULOSIS

IN the July issue of the *American Review of Tuberculosis*, Krause, in one of his engaging and sensible papers, specifies and emphasizes anew the place of laboratory aids in the diagnosis of tuberculous infections. He shows that a thorough-going sputum examination and an intelligently performed tuberculin test are of great value to the diagnostician and more especially to the patient. What is more, from his long, wide and rich experience in the laboratory and in the clinic he is able to tell us precisely what constitutes a thorough-going sputum examination and the manner in which a tuberculin test should be done, and what he says can be read with profit by the practicising physician. The bacteriologic examination of the sputum, the urine, the spinal fluid with a positive result may help to establish a diagnosis before the clinician is able to locate or map the lesion, while a negative result may supply one more point in a diagnosis by elimination. But, such examinations are too often left to an insufficiently trained, impatient examiner and too often, even under better circumstances, this important help is skimped. The collection of the specimen, the type of container, its trip to the laboratory are details that are not to be slighted if the search for tubercle bacilli is to be profitable. The bacteriologist fails in his rôle unless he brings to his aid all the methods devised for the detection of sparse tubercle bacilli. Krause tells about the portions of the specimen to be examined and how the hunt should be made. The failure to find bacilli, even after a long and patient microscopic search, taken alone, does not justify a negative report. There are the concentration and cultural methods, and the animal inoculations that are to be employed before one is warranted in saying that the specimen contains no tubercle bacilli. Corper, in the August 11th issue of the *Journal of the American Medical Association* gives the details for finding tubercle bacilli by an important isolation technic, leading to what he calls "Certified diagnosis of tuberculosis." The finding of tubercle bacilli means the presence of a tuberculous process. The failure to find them may mean the absence of such infection. In either case the report is only of value when the examination is properly made and it is this "properly made" that Krause describes and stresses.

We wonder how many physicians employ the tuberculin test on patients coming to them with ailments of obscure origin or with symptoms suggesting tuberculous infection, and of those who do employ the test we wonder how many know whether or not the tuberculin they are using is active or of required potency. As Krause says, physicians have been misled by reports of European observers on the universality of tubercle infection, and reasoning further that a positive tuberculin reaction fails to differentiate between latent and active tuberculosis, have discounted its value and largely discontinued its use. In the first place, tuberculous infection is not as common in this country as it is or was in Europe, and it is steadily becoming less common. This fact has been convincingly shown by Chadwick, who has applied the von Pirquet test to thousands of Massachusetts school children. This means that a negative reaction to tuberculin indicates the absence of tuberculous foci and this disease may be eliminated from diagnostic possibilities. In the second place, as tuberculosis morbidity declines childhood and latent infection decreases, so the tuberculin test should increase in value. A tuberculin test, however, is not so simple an affair that it can be entrusted to careless, inexpert hands. Its worth makes it worthy the physician's close and personal attention. Not all tuberculins are reliable—some are weak and some have been found to be actually inert. The von Pirquet test must be nicely done and before tuberculosis is excluded an intradermic or Mantoux test should be performed. Here, especially, the results are only as valuable as the perfection of the technic, but there are no difficulties or intricacies that should daunt the average practitioner of medicine.

Krause is quite as aware of the limitations of

laboratory aids to the diagnosis of tuberculosis as he is of the elsewhere unappreciated value of sputum examination and tuberculin testing. As he says, "no laboratory procedure can at present carry the whole load in the positive diagnosis of tuberculosis," and again, "the diagnosis of pulmonary tuberculosis must be, in actual practice, a diagnosis by elimination, by exclusion." We would add that the value of a diagnosis in this disease varies directly with its nearness to the onset of symptoms. Early diagnosis means everything to the tuberculous. Therefore, it would seem that the neglect of any aid to diagnosis is unfair to the patient and that because of the importance of sputum examination and the tuberculin test, not only should they be employed at the beginning in every doubtful case but their method and their practice should be made the personal concern of the physician.

NEW YORK'S SANITARY CODE

THE new sanitary code established by the Public Health Council of New York went into effect on June 30, having been prepared for by the milk producers and handlers of the State for nearly a year. According to the new requirements no person shall sell milk or cream except that which is to be consumed on the premises where sold, without a permit from the health officers. Permits, moreover, will be issued only to those who conform to the code, an essential feature of which is that holders of permits must at all times have a complete record of the persons from whom they purchase milk, and any change in the milk supply must be made known to the health officer.

Rigid inspection of all milk plants and places of selling must be permitted by the holder, and inspection of every dairy farm, with bacterial examination of the milk, is required of the health officer before the permit is issued. The code further contains provisions regarding the production of the milk and its bottling and pasteurization. Milk is classified and must be labelled with one only of the following designations: Pasteurized Grade A, certified pasteurized Grade B, raw Grade A and Grade B.

Stringent regulations apply to the health of milk handlers, who must be examined by the health officer before being allowed to engage in their duties, and all cases of illness on a dairy farm must be reported to the health officer by the physician in attendance. No milk or cream can be shipped into the State unless the receiver or distributor shall have furnished evidence that it was produced under conditions equal to the requirements of the State.

Most large cities have adequate supervision of their milk supplies, and epidemics of disease due to milk are now extremely rare in modern metropolitan centres. It is in the smaller towns and in rural districts that milk is most frequently a carrier of disease, and for such localities State supervision is essential if proper regulations are to be enforced. Recently the town of Lee, in Massachusetts, had an experience with septic sore throat that should furnish a lesson to all such communities. Last year the town of Lincoln indulged in a disgraceful episode with milk borne typhoid fever which should be chalked up against the local, and purely local, supervision of milk production.

The dweller in the country, equally with the city dweller, is entitled to the common decencies of civilization as regards the protection of his health. Until the State Department of Public Health is entitled to step in and guarantee him these decencies, such experiences as those of Lincoln and Lee and many others will be repeated.

Our legislative bodies have never been backward in the framing of new laws. Like suckers, one is born every minute. Occasionally, however, they should indulge themselves to the extent of passing laws of universal value, such as one which would require the State regulation of all milk sold in the Commonwealth, including its universal pasteurization.

THE CANCER CAMPAIGN

THIS week's issue of the JOURNAL carries a report of the cancer campaign in Massachusetts, which was conducted the last week in April through the united efforts of the organized public, the organized profession and organized government. All methods of informing the public and the profession were used and eminent physicians from without the State gave generously of their time. Official recognition of the importance of the subject was given by the attendance at the meetings of Governor Fuller and the mayors of the various cities. Well over 10,000 persons were brought under medical care as an immediate result and the more remote result of the education can never be estimated.

We have recently expressed our utter lack of sympathy with the criticism that the campaign created phobia. That this was not true is shown by the fact that although the attendance at the clinics doubled during and immediately after the campaign, the proportion of those coming with cancer remained about constant (26 to 27 per cent.). Falsehoods, not facts, inspire phobias.

It was perhaps particularly fortunate at this time to make a public attack on cancer. Not long since considerable criticism was heard as to professional lethargy in attacking this disease and cancer legislation was passed. Now the State Department of Public Health and the medical profession have shown forcibly that they have kept faith with the mandate of the Legislature in this regard.

For those who look askance at clinics as draining their offices it is pertinent to note that the attendance at private offices was largest in the cities having clinics, probably because of the more intensive publicity focused about these

clinics. Also, for every person visiting a clinic there were apparently twenty-two who went to private offices.

But what of the future? Such campaigns must be infrequent events to be effective. We hear of service in other fields dying of dry rot; of the weekly tuberculosis clinic in a neighboring city not visited by a physician for six weeks; of the venereal disease clinic where the tourniquet is left in place during intravenous therapy! Shall the cancer service so deteriorate as our interest is distracted? From time to time the JOURNAL carries annual summaries of local cancer clinics. They are a tribute to a few sincere, personally disinterested physicians and lay people. From time to time through interchange of consultants, special clinics, meetings, and the like, local interest must be stirred and appreciation maintained. Above all, each practising physician must remember that in all this cancer activity resulting from a public demand made articulate by the Legislature, a great faith in the medical profession has been expressed. This faith must not be abused!

WHO OUGHT TO REPRESENT THE TWELFTH DISTRICT IN CONGRESS?

THE JOURNAL is not ordinarily concerned with politics. But it is proper to publish the record upon matters of public health of a candidate for Legislative office.

State Senator William I. Hennessey of Dorchester is seeking to win the Democratic nomination in the 12th Congressional District. For many years this District has been ably represented by the late Congressman Gallivan, who with his brother, the late Dr. Gallivan, always worked for the best possible public health laws. Ordinarily the Democratic nomination is equivalent to an election in this District.

Senator Hennessey has been an active opponent of vaccination year after year in the Massachusetts Legislature and a year ago voted in favor of the establishment of a special board of Chiropractic examiners. This latter vote was cast after conference with certain physicians in his District at which time the need of adequate preparation for the practice of the healing art was explained to him and at which time these physicians understood he would vote to maintain the single standard now in force.

Unless the physicians of South Boston and Dorchester are willing to be represented in Washington by an aggressive opponent of measures for the betterment of health, they must exert their power to defeat the nomination in the September primaries of State Senator William I. Hennessey.

The voters of the twelfth Congressional District must decide who will be best qualified to represent them.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ALBEE, GEORGE M. M.D. N. Y. University, 1893. Cardiologist, Worcester City Hospital. Consulting Cardiologist, Fairlawn and Louis Pasteur Hospitals, Worcester. His subject is: "The Care of the Heart in Pneumonia". Page 399. Address: 390 Main Street, Worcester, Mass.

ADAMS, F. DENNETTE. Litt. B., M.D. Harvard 1917. Physician to Out-Patients, Massachusetts General Hospital; Assistant in Medicine, Harvard Medical School. His subject is: "Some Clinical Observations on Lobar Pneumonia". Page 402. Address: 226 Marlboro Street, Boston.

BINNEY, HORACE. A.B., M.D. Harvard 1901; F.A.C.S. Surgeon-in-Chief, Boston City Hospital. Consulting Surgeon, Malden Hospital. Professor of Surgery, Tufts College Medical School. His subject is: "The Treatment of Empyema Following Pneumonia". Page 410. Address: 403 Beacon Street, Boston.

CECIL, RUSSELL L. A.B., Sc.D., M.D. Medical College of Virginia 1906. Assistant Visiting Physician, Bellevue Hospital, N. Y. City. Assistant Professor of Clinical Medicine, Cornell University Medical School, N. Y. City. His subject is: "The Recent Advances in the Specific Treatment of Pneumonia". Page 415. Address: 31 East 61st Street, New York, N. Y.

MEAKER, SAMUEL R. A.B., M.D. Harvard 1915, M.R.C.S. England, F.A.C.S. Associate Professor of Gynecology, Boston University School of Medicine. His subject is: "Posterior Median Discission of the Cervix". Page 427. Address: 475 Commonwealth Avenue, Boston.

CLINE, SAMUEL. S.B., M.D. Harvard 1915. Laryngologist, Boston Sanatorium; Clinical Assistant in Otolaryngology, Massachusetts Eye and Ear Infirmary; Assistant Surgeon in Otolaryngology, Children's Hospital; Assistant Laryngologist, Beth Israel Hospital. His subject is: "Laryngeal Tuberculosis". Page 429. Address: 353 Commonwealth Avenue, Boston.

BALCH, FRANKLIN G. M.A., M.D. Harvard 1892, F.A.C.S. Consulting Surgeon Massachusetts General Hospital, Boston; Lawrence Memorial Hospital, Medford; Surgeon, Faulkner Hospital, Jamaica Plain. Address: 279 Clarendon Street, Boston. Associated with him are:

BIGELOW, GEORGE H. A.B., M.D. Harvard Medical School 1916, Doctor of Public Health 1921, Commissioner of Public Health of Massachusetts. Address: Department of Health, State House, Boston, Mass. And

GREENOUGH, ROBERT B. A.B., M.D. Harvard 1896, F.A.C.S. Visiting Surgeon Massachusetts

General Hospital; Assistant Professor Surgery, Medical School, Harvard University; Director Cancer Commission of Harvard University; Surgeon in Charge, Collis P. Huntington Memorial Hospital. Address: 8 Marlboro Street, Boston. They write on "Results of the Massachusetts Cancer Campaign". Page 430.

BOSTON MEDICAL LIBRARY

Treatment of General Paresis by Malarial Plasmodia

The human organism cannot often permit itself to become the battle ground of enemies mutually antagonistic and not suffer as much, if not more, than the combatants themselves. The introduction into present day therapy of the malarial organism for the purpose of overcoming or counteracting the effects of general paresis is an instance in point and recalls the story of the importation of the English sparrow to exterminate the worms that were destroying the English ivy on the walls of Boston's houses. A celebrated Boston wit who was asked which he regarded as the more undesirable the "worms or the sparrows," replied that he did not know as he never had had the sparrows, an answer that might conceivably be offered by one who had had paresis but never experienced the agonies of quartan or tertian ague. It has doubtless fallen to the lot of many to observe an abatement of symptoms in certain toxic diseases when an intercurrent infection has been superimposed, e. g., a toxic arthritis has lost its characteristic joint symptoms in the presence of a severe lobar pneumonia. Many observations of a like nature have been made and recorded. So well established has become the causal relation of syphilis to paresis that the lesions of the latter may well be regarded as having a toxic origin. It was left for Noguchi and Moore to positively establish this relationship by identifying the spirochetes in the cerebral tissues and to discover chemical, serological and cytological data which are of great service in the laboratory study of this disease. In 1581 Willis first recognized paresis as an entity. Columbus's sailors are credited with having brought syphilis back to Europe from America, in 1498, so that this recognition of the cerebral type of syphilitic lesions followed, with reasonable promptness, upon the introduction of the responsible infection.

The cerebral lesion is an inflammation in the arachnoid membrane. This also was demonstrated by Willis. In 1798 John Haslam of Edinburgh reported the first case in literature, before the Edinburgh Royal Medical Society. Bayle, in 1822, gave the first accurate description of the pathologic anatomy of general paresis. As early as 1816 it had been noted that remissions of paretic symptoms occurred in the midst of attacks of certain acutely febrile diseases. Knowledge of this led Wagner and Jouregg in 1917 to inoculate a patient who suffered from paresis with blood drawn from a malarial subject. About twenty-eight days later the patient had his first chill; he was allowed to have twelve others before his malaria was treated with quinine. The apparent effect is seen in making the remission of symptoms, which is more or less characteristic of the disease, occur with greater frequency and have a longer duration.

Eldredge, who reports a series of cases in the *Journal of the American Medical Association* Vol. 84; 1097-1101, Apr. 1925), concludes that the method gives the greatest promise of prolonged remissions. His results, represented in percentages, indicate slightly better than 70% either improved or arrested. Just what sort of reaction it is that selects one of the normal phases of a disease and draws it out, it is difficult to understand. Malarial organisms must have some inhibitory effect upon the production of whatever toxic material the spirochetes manufacture and so the actively progressive phase of the disease is delayed.

The literature has been very abundant since 1921, and during the fortnight beginning with September 4, some of the most important articles will be displayed in Holmes Hall.

MISCELLANY

A VISIT TO THE PRENDERGAST PREVENTORIUM

On August 16, inst. by the courtesy of Dr. John B. Hawes, 2nd, President of the Boston Tuberculosis Association and Miss Bernice W. Billings, Executive Secretary, we together with Mr. H. Morison, a member of the Executive Committee of the Boston Health League, were privileged to visit the Preventorium.

Beginning several years ago as a summer camp in an inadequate building on undeveloped grounds it was a pleasant surprise to find that the Preventorium had grown to the extent of occupying a beautiful structure costing ninety-two thousand dollars with about one hundred boys and girls as guests. This organization may be regarded as the major function of the Boston Tuberculosis Association for which it appropriated about fifteen thousand dollars for maintenance this year and expects to spend at least twenty-five thousand dollars next year.

The children are between the ages of five and twelve years and are all contact cases. They are received from families in Boston from which a case of tuberculosis has been reported and in addition to the fact of being classified as contacts each one has a positive von Pirquet. The children are admitted the latter part of June and stay until the last of August. There will be a full year class of about forty selected cases. The appearance of the children is excellent physically and the behavior equal to that expected of well-trained boys and girls of that period of life. The senior councillors are young women college graduates and some of the junior councillors are recent graduates of the Pre-

ventorium. The daily routine is as follows:

DAILY SCHEDULE

6:45	Rising Bell
7:00	Flag Raising
7:15	Setting-up exercises
7:30	Breakfast
8:15	Morning duties
8:45	Inspection
9:00-11:00	Recreation
11:00	Rest hour
11:45	Wash-up
12:00	Dinner
1:00-3:00	Rest
3:00	Temperatures
3:30-5:20	Recreation
5:20	Wash-up
5:30	Supper
6:00-7:00	Quiet games
7:00	Showers
8:00	Bed

The children are divided into groups for the different exercises and instruction as follows:

Group

9:00	Swimming
9:30	Playground
10:00	Posture
4:00	Scouting
4:45	Nature

Group

9:00	Playground
9:30	Swimming
10:00	Scouting
4:00	Nature
4:45	Bows and Arrows

Group

9:00	Playground
10:00	Swimming
4:00	Letters
4:45	Nature

Group

9:00	Playground
10:00	Nature
10:30	Swimming
4:00	Bows and Arrows
4:45	Posture

ROTATED
Folk dances and quiet games 6-7 P. M.

The future plans for maintenance and development are contingent on financial contributions because the Association is not endowed and the revenue from the seal sales will not be sufficient.

The capital assets of the Boston Tuberculosis Association consist of faith in the generosity of the people of Boston and not in an established endowment. This Preventorium is built on the belief that when a worth-while project has been demonstrated it will not be allowed to die for want of material support. Already several organizations are contributing help in the way of supplies of various kinds and it is reasonable to expect that the City of Boston will come to see the great benefits to the people of this section in the work for the prevention of tuberculosis which also means the prevention of many other health disasters. If children can be made resistant to infections many will live to fill useful places in society who otherwise would become burdens or die. If the city of Boston can be induced to pay one dollar and thirty-five cents a week for the care of these children the Preventorium can be carried on without the constant worry incident to meeting deficits.

In order to supplement the work of the Preventorium a follow-up system is carried on by a very high-grade and well-trained woman in the homes of the children. This domicilliary service is one of the more-important functions of the Association for it insures continued observance of the rules of health taught the children while in the Preventorium.

In addition to doing a great work for the children, the children are lending themselves as material for studies which may contribute very much to the knowledge of tuberculosis. The findings of physical and X-ray examinations are being correlated and studied by clinicians and technicians at first independently and later at conference with the expectation of ascertaining the exact meaning of physical signs and technical findings. It is reasonable to hope that the Prendergast Preventorium is to give information which will be a distinct contribution to medicine.

If anyone will devote time to a visit to the Preventorium under the guidance of Miss Billings he will find much of interest and will be impressed with the unusual ability of the Secretary of the Boston Tuberculosis Association in promoting the efficient administration of that institution.

THE COST OF ILLNESS

Homer Folks, Secretary of the State Charities Aid Association of New York, in an address delivered before the International Conference of Social Work at Paris reported that he estimated the cost of illness in the United States to be $134.68 per family or $31.08 per capita per year. He estimated that physicians received $745,000,000 and quacks $120,000,000; hospitals were paid $404,501,572 and medicine and supplies cost $700,000,000. The loss of wages through illness is estimated to be $1,245,000,000.

This tremendous amount can be reduced when more money is spent for public health work and the application of better understanding of the fundamental causes of illness.

DEFECTS IN CHILDREN

PHYSICAL DEFECTS IN CLEVELAND SCHOOL CHILDREN

The bureau of physical welfare of the Cleveland Board of Education reports that examination of nearly 73,000 school children from the kindergarten and early school grades showed that at least 7 out of every 10 had physical defects. Nearly one-sixth were suffering from malnutrition, largely because of wrong choice of food rather than poverty; 32,000 children had defective teeth, 7,000 had goiter, 1,600 had heart disease, and 2,000 had defective hearing. Nearly 20,000 children of 14 to 18 years who applied for work permits showed, in general, a still larger percentage of defects. Since many of the physical defects can be prevented or controlled the department of health education believes that the necessity for more health work in schools is clearly demonstrated.—*Children's Bureau, Washington.*

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1927 AND SEVEN YEAR AVERAGE

MONTH ENDING AUGUST 4

1928	Wk. ending July 14	Wk. ending July 21	Wk. ending July 28	Wk. ending Aug. 4	Average cases reported for week corresponding to Aug 4 for past seven yrs.	Wk. ending July 16	Wk. ending July 23	Wk. ending July 30	1927 Wk. ending Aug. 6
Actinomycosis	-	-	-	-	-	-	-	-	-
Anthrax	-	-	-	-	-	-	-	-	-
Botulism	-	-	-	-	-	-	-	-	-
Cerebrospinal Men.	1	-	1	1	-	2	1	-	-
Chickenpox	19	8	5	10	7	45	30	33	21
Conjunctivitis Inf.	-	-	-	-	-	-	-	-	-
Diphtheria	17	13	9	16	20	22	16	23	17
Dysentery, Bacillary	-	-	-	-	-	-	-	-	-
Dysentery, Amoebic	-	1	-	-	-	-	-	-	-
Encephalitis, Epid.	2	-	-	-	-	-	-	-	-
Favus	-	-	-	-	-	-	-	-	-
German Measles	7	2	1	-	1	-	2	-	-
Hookworm Infection	-	-	-	-	-	-	-	-	-
Influenza	1	1	3	4	2	-	2	3	1
Leprosy	-	-	-	-	-	-	-	-	-
Malaria	-	-	1	-	1	-	-	-	3
Measles	192	148	109	67	28	41	21	10	19
Mumps	31	27	12	10	5	21	6	6	14
Paratyphoid Fever	-	1	-	-	-	-	-	-	-
Pneumonia (Broncho)	6	18	15	16	6*	15	15	8	9
Pneumonia (Lobar)	10	14	7	6	5	12	10	5	8
Poliomyelitis	-	3	1	3	5	3	-	1	11
Scarlet Fever	24	14	5	10	15	18	11	10	10
Septic Sore Throat	9	3	1	5	1	-	-	-	2
Smallpox	-	-	-	-	-	-	-	-	-
Tetanus	1	-	1	-	-	1	1	1	-
Trachoma	-	-	-	-	-	-	-	-	-
Trichinosis	-	-	-	-	-	-	-	-	-
Tuberculosis (pul.)	33	38	28	39	28	55	35	85	30
Tuberculosis (o.f.)	1	8	3	7	4	3	1	4	7
Typhoid Fever	1	2	1	-	9	3	2	3	1
Typhus Fever	-	-	-	-	-	-	-	-	-
Undulant Fever	-	-	1	-	-	-	-	-	-
Whooping Cough	87	56	73	87	37	32	27	26	47
Gonorrhoea	29	25	60	31	20	12	18	26	40
Syphilis	29	25	50	46	25	10	9	37	81

*Average for three years. Made reportable January 1,1925. Remarks: No cases of cholera, Asiatic, glanders, plague, rabies in humans and yellow fever during the past seven years.

LEGISLATIVE MATTERS

COMPULSORY VACCINATION FOR ALL SCHOOL CHILDREN

Year after year the matter of the compulsory vaccination of children attending private as well as of children attending public schools has been discussed in the Legislature.

The Medical Societies of the State Chambers of Commerce, heads of private schools, the Presidents of practically every College in the State, Boards of Health and other civic organizations have favored doing away with an existing discrimination.

They favor the Washburn Bill which will be presented to the Legislature of 1929 because they believe that if enacted it will be an added safeguard to the public health and will prevent a group of individuals growing up who may at any time become a menace to the community.

The law now provides that a child may be exempt if a physician certifies that for any cause his health would be injured by vaccination. The bill provides that the reason for the exemption must be stated in the certificate and that the certificate shall be good only for one year.

In the Session of 1927 this Bill was passed by the House. The Senate defeated it.

In September at the primaries candidates for the Senate and House are nominated for election in November.

The Bill is opposed by a small but very active group of citizens. In the past members of the Legislature have stated that the merits of the bill were never explained to them and that they pledged themselves to oppose it without hearing from those who favored it.

The members of the Auxiliary Committee on Legislation have been asked to interview candidates for the Senatorial nominations. Every reader of the JOURNAL is urged to aid by interviewing these candidates, who in general are more receptive to suggestions and arguments before rather than after election.

We present herewith the published list of Senatorial candidates to be voted for in the primaries:

REPUBLICANS	DEMOCRATS
Cape and Plymouth Donald W. Nickerson	Cape and Plymouth—
Bristol No. 1— William J. Davison, Taunton James G. Moran, Mansfield	Bristol No. 1— James P. Lamont, Taunton
Bristol No. 2— James H. Kay, Fall River Walter E. McLane, Fall River	Bristol No. 2— William S. Conroy, Fall River
Bristol No. 3— Alfred M. Bessette, New Bedford Nathaniel P. Sowle, New Bedford	Bristol No. 3— Charles L. Booth, New Bedford
Plymouth— William B. Baldwin, Brockton Roger Keith, Brockton Frank W. Eaton, Brockton	Plymouth— Joseph La Fontaine, Brockton
Norfolk and Plymouth— Henry F. Ripley, Hingham David Perry Rice, Rockland George A. Dennett, Sharon	Norfolk and Plymouth— John H. Moran, Weymouth
Norfolk— Henry L. Kincaide, Quincy	Norfolk—
Norfolk and Middlesex— Samuel H. Wragg, Needham George F. James, Norwood	Norfolk and Middlesex—
Norfolk and Suffolk— Erland F. Fish, Brookline Frederic E. Dowling, Boston Joseph A. Conway, Boston Bertha Cook, Boston	Norfolk and Suffolk— Daniel F. O'Connell, Brookline
Suffolk No. 1— Conde J. Brodbine, Revere John E. Beck, Chelsea Edward J. Cox, Chelsea	Suffolk No. 1— Hugh F. McCarthy, Chelsea
Suffolk No. 2— Lamy Rivoire, Boston	Suffolk No. 2— John B. Cashman, Boston William J. Francis, Boston John P. Buckley, Boston Daniel J. Murphy, Chelsea Robert Dinsmore, Boston Joseph A. Langone, Jr., Boston
Suffolk No. 3— Henry Parkman, Jr.	Suffolk No. 3— Frank J. Lehan, Cambridge Michael J. Hurley, Boston Edward J. Murphy, Cambridge Daniel L. Herlihy, Cambridge
Suffolk No. 4— Mary M. Riley, Boston	Suffolk No. 4— James E. McConnell, Boston Robert E. Bigney, Boston
Suffolk No. 5— John M. Brennen, Boston	Suffolk No. 5— Hugh J. Campbell, Boston Michael J. Ward, Boston Stephen R. Mealey, Boston
Suffolk No. 6— Gaspar G. Bacon, Boston James F. Hanley, Boston	Suffolk No. 6— William H. Powderly, Boston Arthur J. Coughlin, Boston Nathaniel M. Clark, Boston Bernard C. Dwyer, Boston
Suffolk No. 7— Frederick E. Hawley, Boston	Suffolk No. 7— James J. Moynihan, Boston Richard J. Garvey, Boston Joseph J. Mulhern, Boston
Suffolk No. 8— Thomas H. Bilodeau, Boston Walter E. Jackson, Boston Max Ulin, Boston Frank H. Luce, Boston	Suffolk No. 8— John T. Blong, Boston John F. Buckley, Boston Robert Robinson, Boston
Essex No. 1— Charles H. Annis, Lynn Frank W. Osborne, Lynn Charles C. O'Donnell, Lynn James D. Bentley, Swampscott	Essex No. 1— Joseph H. Haggerty, Lynn Charles C. O'Donnell, Lynn William F. Shanahan, Swampscott
Essex No. 2— Willard O. Wylie, Beverly James A. Torrey, Beverly	Essex No. 2— James D. Burns, Salem
Essex No. 3— Cornelius F. Haley, Rowley	Essex No. 3—
Essex No. 4— George Ross French, Salisbury J. Bradford Davis, Haverhill	Essex No. 4— Patrick J. Doran, Amesbury
Essex No. 5—	Essex No. 5— Denis W. Delaney, Lawrence James E. Warren, Lawrence John T. Coan, Lawrence
Middlesex No. 1— Harold William McKelvey, Wayland Arthur W. Hollis, Newton	Middlesex No. 1— Robert T. Allen, Newton Edward J. Carey, Framingham
Middlesex No. 2— Clarence P. Kidder, Cambridge	Middlesex No. 2— Joseph W. Monahan, Belmont
Middlesex No. 3— Warren C. Daggett, Somerville Lyman A. Hodgdon, Somerville	Middlesex No. 3— Douglass B. Foster, Somerville Maurice F. Ahearn, Somerville
Middlesex No. 4— Angier L. Goodwin, Melrose Alvin E. Bliss, Malden	Middlesex No. 4—

Middlesex No. 5— George G. Moyse, Waltham Henry T. G. Dyson, Hudson	Middlesex No. 5— Paul A. Mogan, Waltham
Middlesex No. 6— Lewis Hilton Peters, Medford Charles C. Warren, Arlington	Middlesex No. 6— Charles T. Daly, Medford Charles A. Coughlin, Medford
Middlesex No. 7— Joseph R. Cotton, Lexington Alice F. D. Pearson, Lowell	Middlesex No. 7—
Middlesex No. 8— Walter Perham, Chelmsford Edward Gaston Campbell, Lowell	Middlesex No. 8— Michael H. Hoar, Lowell
Worcester No. 1— Christian Nelson, Worcester Charles Ward Johnson, Worcester Albert A. Wheeler, Worcester	Worcester No. 1— Thomas E. Dowd, Worcester
Worcester No. 2— George H. Nelson, Worcester Charles S. Holden, Worcester	Worcester No. 2— James J. McGrail, Worcester Charles E. Scott, Worcester
Worcester No. 3— Thomas J. Worrell, Athol	Worcester No. 3— John H. Weaver, Worcester
Worcester No. 4— Elbert M. Crockett, Milford	Worcester No. 4—
Worcester and Hampden— Thomas H. Johnston, Clinton Warren E. Tarbell, East Brookfield Clifford F. Chamberlain, Sturbridge	Worcester and Hampden— Andrew J. Leach, Brookfield
Berkshire— J. Arthur Baker, Pittsfield Harvey A. Gallup, North Adams	Berkshire—
Berkshire, Hampshire and Hampden— Frederick E. Judge, Southampton Harry B. Putnam, Westfield William L. Dubois, West Stockbridge	Berkshire, Hampshire and Hampden— Henry W. Warner, Northampton
Franklin and Hampshire— Cady R. Elder, Amherst	Franklin and Hampshire—
Hampden No. 1— C. Wesley Hale, Springfield	Hampden No. 1— John S. Kane, Springfield
Hampden No. 2— Walter M. Grocli, Chicopee John F. Shea, Holyoke	Hampden No. 2— David F. Sullivan, Holyoke James F. Sweeney, Holyoke Patrick F. Monahan, Holyoke

CORRESPONDENCE

A CORRECTION OF DR. COTTON'S STATEMENT

To the Editor of the
NEW ENGLAND JOURNAL OF MEDICINE.

Dear Sir:

In Dr. Cotton's letter in your issue of August 23, 1928, relating to criticism of the Board of Registration in Medicine he says in part: "Most of the criticism has been to the effect that the Board has committed itself to price fixing for medical services. Nothing, I think, could be further from the fact, and I say this after having talked with members of the Board."

Surely there must be misinformation, misunderstanding, or confusion on this subject; the files of the Board of Registration show that the findings in the Henderson case on which were based the punishment meted by the Board to Dr. Henderson were: (1) Gross misconduct in the professional care of a patient. (2) Extortion in the matter of a fee. On Dr. Henderson's appeal to the Supreme Court, Judge Carroll's ruling was apparently based solely on the size of the fee. These facts have come to my attention in the course of an investigation on behalf of the Committee on Ethics and Discipline of the Massachusetts Medical Society.

Very truly,
DAVID CHEEVER.

ANTI-RABIC TREATMENT SHOULD BE PROVIDED FOR ALL NEEDY PERSONS

August 20, 1928.

Dear Mr. Editor:

Already in the JOURNAL there has appeared some comment on the cost of the Pasteur anti-rabic treatment and I am now using its pages to express the opinion that the State should make provision for supplying this treatment to those with little money.

In the early months of the year a dog, that had been handled intimately in the laboratories of the Harvard Medical School by an assistant in the Department of Medicine and a laboratory chemist, developed rabies. There was a risk that these two men had become infected. The obvious thing to do was to give them the Pasteur anti-rabic treatment.

Much to my surprise I discovered then that the cost would be $25.00 per man and that neither the City of Boston nor the Commonwealth of Massachusetts had made any provision to supply such treatment free of cost to those unable to pay for it. To me this seemed a deficiency in the Board of Health of one or both.

The Commonwealth of Massachusetts does prepare and distribute various preventive and curative sera. In my opinion this should be done for the Pasteur anti-rabic treatment which is so effective in preventing the most horrible form of death that the modern medical man witnesses.

I hope that our Commissioner of Health, Dr. Bigelow, will use every effort possible to obtain promptly such legislation and appropriation as is required to make possible the distribution to any needy victim of the bite of a rabid dog the Pasteur anti-rabic treatment. I sincerely hope that the medical profession of Massachusetts will support him strongly in such an effort.

Yours truly,
HENRY A. CHRISTIAN,
Physician-in-Chief, Peter Bent Brigham Hospital, and Hersey Professor of the Theory and Practice of Physic, Harvard Medical School.

NEWS ITEMS

DR. RICHARD M. SMITH RECEIVES DEGREE

Dr. Richard M. Smith, of Boston, had this year the honorary degree of Doctor of Science conferred on him by Williams College, his alma mater. This degree, unusual as it is for a clinician to receive, is nevertheless well merited, for both as a practitioner of pediatrics and as assistant professor of hygiene at the Harvard School of Public Health his name has received nation-wide recognition.

Dr. Smith's medical career began when he first became an assistant to Dr. John Lovett Morse, and

his own ability soon caused him to become recognized as a leader in his specialty. The science of child hygiene has owed its growth largely to his activities, and he has always been in the front rank of those who have contributed to the development of infant hygiene in Boston. The JOURNAL joins with his many friends in congratulating Dr. Smith on his new honor.

DR. F. D. DONOGHUE GOES ABROAD—Dr. Francis D. Donoghue sailed on the S. S. Ile De France on Friday, August 17th, on his way to attend the Fiftieth International Congress on Industrial and Occupational Diseases which will open in Budapest September third and continue until September eighth.

DR. C. P. SYLVESTER SAILS FOR PARIS—Dr. C. P. Sylvester, Chairman of the Board of Registration in Medicine, sailed August 17th to visit Mrs. Sylvester in Paris. He will make only a short visit abroad.

NOTICE

CORRECTION

The paper of Dr. Adolf Meyer entitled "The 'Complaint' as the Center of Genetic-Dynamic and Nosological Teaching in Psychiatry" which appeared in the issue of August 23 was read before the Massachusetts Psychiatric Society instead of the New England Psychiatric Society as previously stated.

NOTICE OF MEETING

AMERICAN ELECTROTHERAPEUTIC ASSOCIATION

The Thirty-eighth Annual Meeting of the American Electrotherapeutic Association is to be held September 10th-14th, at the Hotel Statler, Boston, Massachusetts. The program of the meeting includes papers and Committee Reports regarding all phases of Physical Therapy, both from the standpoint of research as well as of clinical application. Two days will be devoted to visiting physical therapy clinics in Boston institutions and also to a visit to the plant of the General Electric Company in West Lynn. There will be also a large technical exhibit demonstrating new apparatus and accessories. All legally licensed physicians are welcome, and programs can be obtained by addressing the Secretary, Dr. Richard Kovacs, 223 East 68th Street, New York.

SOCIETY MEETINGS

September 10-14—American Electrotherapeutic Association. Complete notice appears above.

September 18, 19, 20—Fourth Clinical Congress, Connecticut State Medical Society. For detailed notice see page 394, issue of August 23.

September 24-28—Sixth Conference of the International Union Against Tuberculosis. For complete notice see page 349, issue of August 16.

October 1-12—The Graduate Fortnight of the New York Academy of Medicine. For complete notice see page 396, issue of August 23.

October 15-19—Interstate Post Graduate Association. Complete notice on page 1025, issue of June 28.

December 3-7—Radiological Society Convention. Detailed notice appears on page 712, issue of May 17.

DISTRICT MEDICAL SOCIETIES

Barnstable District Medical Society

Schedule of Meetings: November 1, 1928, February 7, 1929, May 2, 1929.

Berkshire District Medical Society

The especial features of the October meeting will be addresses by Dr. George Crile of Cleveland, Dr. Emil Novak of Johns Hopkins, Dr. Willis Campbell of Memphis, Tennessee, and Dr. Thomas Ordway of Albany, N. Y. A complete program will be published later.

HUGH J. DOWNEY, Secretary.

Bristol North District Medical Society

September 21—Fall meeting. Place to be determined later.

April 18, 1929—Spring meeting.

Bristol South District Medical Society

Meetings will be held November 7, 1928, and May 2, 1929. Complete notice will appear later.

Essex North District Medical Society

October, 1928 (Wednesday afternoon)—A joint meeting with Middlesex East and North, and Essex South. Date and place to be announced.

November 1, 1928 (Thursday)—Censors meet at Hotel Bartlett, 95 Main Street, Haverhill (Tel. 3430), at 2 P. M.

January 2, 1929 (Wednesday)—Semi-annual meeting at Haverhill, at 12:30 P. M.

May 1, 1929 (Wednesday)—Annual meeting at Lawrence, at 12:30 P. M.

May 2, 1929 (Thursday)—Censors meet at Hotel Bartlett, 95 Main Street, Haverhill, at 2 P. M. (Tel. 3430.)

Middlesex East District Medical Society

The following schedule of meetings has been arranged for the coming year:

September 12—Winchester Country Club.
October 17—Middleton. (Joint meeting with Essex North and South and Middlesex North.)
November 14—Woburn.
January 16—Wakefield.
March—Reading.
May—Melrose.

Subjects and speakers to be arranged by committees in each city or town.

Middlesex North Medical Society

October 17—A combined meeting with Essex North and South.

The winter meeting will be held the last Wednesday in January.

The annual meeting will be held the last Wednesday in April.

Norfolk District Medical Society

Below are the proposed meetings of the Norfolk District Medical Society for the 1928-29 Season. The schedule below will be adhered to as strictly as possible. The carrying out of the proposed plan will, of course, depend upon the acceptances of invitations extended to the speakers. Minor changes may be necessary.

October 30—Pondville Hospital. Dr. Lyman F. Jones, Superintendent. Inspection, 4 P. M.; Business Meeting at 5:30; Papers by the Staff; Collation.

November 21—Boston Medical Library. In combination with Suffolk District and Middlesex East District Societies. Subject, Physiotherapy—Dr. F. B. Granger.

January 29—Roxbury Masonic Temple. Subject, Treatment of Varicose Veins by the Injection Method—Dr. E. E. O'Neil. Dr. Homans will open the discussion.

February 26—Roxbury Masonic Temple. Dr. W. R. Ohler. Subject to be announced. Discussion to be opened by Dr. A. A. Hornor.

March 26 — Roxbury Masonic Temple. Subject and speakers to be announced.

May, 1929—Annual meeting program to be announced.

The Censors of the Society meet November 8, 1928, and May 9, 1929, at the Roxbury Masonic Temple, Warren Street, Roxbury, at 4 P. M., for the examination of candidates.

BOOK REVIEWS

The Surgical Clinics of North America, June, 1928. Chicago Number.

Under the title of "Gastro-enterostomy Disease" Dr. Arthur Dean Bevan recounts some of his experiences with the complications which may follow gastro-enterostomy. These cases are classified in four general groups; vicious circle with vomiting developing soon after operation; vicious circle coming on weeks or

months after operation; jejunal ulcer; gastro-intestinal invalidism without evidence of organic trouble. He is an advocate of the operation for given indications and states that in general his experience with it has been satisfactory.

The important causes of fistulae after appendix operations are also discussed.

Dr. Kellogg Speed presents an interesting series of cases illustrating the clinical course of acute infections of the capital epiphysis of the femur. In the fulminating cases the differential diagnosis involves many diseases including certain acute abdominal conditions. Early diagnosis and drainage of the hip joint is imperative. The end result of drained and healed acute epiphysitis may be indistinguishable from Legg-Perthes' disease.

Dr. Philip H. Kreuscher describes the technic of incisions for approach to the shoulder, elbow, knee and hip joints together with the general surgical principles to be observed in such incisions.

The causes of failure after bladder neck operations are discussed by Dr. Daniel N. Eisendrath. Prominent among these is the rather general failure to recognize that there are many causes beside prostatic hypertrophy for frequency, dysuria and chronic retention in the male after middle life.

Dr. Golder L. McWhorter discusses some surgical disease of the omentum, particularly inflammatory tumors. He also describes the use of cortical inlay bone grafts stressing the importance of avoiding encircling bone sutures.

Dr. Oscar Nadeau gives a clinical demonstration of the use of the cystoscope in the diagnosis of abdominal disease.

Two newly modified and useful pieces of surgical apparatus are described by Dr. David C. Straus. The first is a walking splint for cases of delayed union and non-union in fractures of the bones of the leg. Its important feature is a leather cuff designed to lace up the back so that it fits the tibia accurately. The second is a portable apparatus for traction on the skin in arm amputations.

A statistical study of 110 cases of carcinoma of the breast is presented by Dr. Paul Oliver. Interesting features are the review of initial signs and symptoms in these cases and a discussion of the clinical and gross pathological signs in benign and malignant tumors of the breast.

A variety of interesting urological conditions are represented in the cases presented by Dr. Robert H. Herbst; pyonephrosis; hypernephroma; linear ulcer of the bladder; double kidney and ureter.

Some problems involved in the repair of large ventral hernias are presented by the clinic of Drs. Edmund Andrews and Warner S. Bump, with particular attention to estimating the operability. A demonstration is given of the use of extra long and wide fascial strips in repair. A case of fatal post-operative alkalosis following gastro-enterostomy is discussed. The authors believe that this condition occurs in less fulminating type in a great many cases following gastro-enterostomy and goes unrecognized.

Ano-rectal disease is the subject from the clinic of Dr. Clement L. Martin. Particular attention is given to adenoma of the rectum especially in the matter of treatment.

Dr. Francis H. Straus presents a case of protracted pain in the leg from obscure cause. There was striking absence of localizing signs. He summarizes the lessons taught by this case of abscess of the femoral condyle as follows:

1. The unreliability of X-ray and anatomic guides in differentiating tuberculous and low grade pyogenic bone abscesses.
2. The occasional value of arthrotomy purely as a diagnostic measure.
3. The value of over-exposure in making some bone lesions more evident in the skiagram.
4. The elaborate approach necessary to reach the medial condyle without traversing the knee joint.

Dr. John D. Koucky has observed repeatedly the value of pain in the upper outer aspect of the thigh as a diagnostic sign in torsion of ovarian cyst. He believes it is present in a great majority if not all cases, sometimes as the presenting symptom, but more often overshadowed by the more severe abdominal pain. It has its origin undoubtedly as a referred pain.

Dr. Albert H. Montgomery describes a successful operation for intussusception suggested by the manner in which nature occasionally cures an apparently hopeless case. He sutured the intussusception ring then performed an anastomosis between the ileum and colon above.

Syphilis. Second Edition with 165 illustrations including 16 figures in colors. By HENRY H. HAZEN, A.M., M.D. The C. V. Mosby Company, St. Louis, Mo. 1928.

Though there have been many changes in this edition it carries out the primary object of the book as stated by Dr. Hazen in the first edition: to give in a comparatively small book an authoritative presentation of the whole field of syphilis. Some chapters have been entirely rewritten while the author has taken over some sections formerly prepared by collaborators. All material has been brought up to date, and a late bibliography added. Besides the inclusion of more illustrations, the titles to some of them have been changed so that "they may preach a sermon and not merely state the name of the condition".

This book has one advantage over others on the same subject in that a large percentage of the illustrations are taken from the colored race where the appearances differ materially from similar conditions in the white race and where diagnosis is often exceptionally difficult.

The International Medical Annual. 46th year. William Wood and Company. New York, 1928.

This book is, as the name implies, a general review of the literature of the previous year. Obviously it makes no pretense to completeness, but points of interest are culled from the enormous literature. The contributors are eminent medical men of England. As would be expected from the calibre of the contributors, the book is extremely well written, interesting, and free from errors. It contains abstracts from the fields of internal medicine, surgery, neurology, and here and there touches upon almost all the specialties, thus adding to its value to the general practitioner. To the abstracts are added comments which are pithy and informative.

There are fairly numerous and excellent illustrations. The type is so small as to preclude any continuous reading. In general one is impressed by the care exercised in producing this annual.